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THE PROBLEM OF CORONARY SCLEROSIS

G. WERLEY, M. D.
EL PASO, TEXAS

Coronary sclerosis differs in no way from arteriosclerosis of the aorta so far as pathology is concerned. But its consequences are vastly different. The coronaries supply the most vital organ in the body. An atheromatous projection of no significance into the aorta might readily occlude the smaller coronary vessel almost entirely. The situation of the coronaries makes them particularly vulnerable. Coming off at the headgate of the arterial system they are subject to the same stress and strain as the aorta itself, to the thrust of systole and the recoil of semilunar closure. The main branches of the coronaries lie on the surface of the heart unsupported by surrounding structures, except the pericardium. These facts enable us to understand why the coronaries are sclerosed twice as often as are the arteries of the brain, and three times as often as those of the kidneys.

The consequences of coronary sclerosis are different than in other arteries, due to certain peculiarities. The blood flow in the larger of these vessels is intermittent[1]. There is stasis in systole and rapid flow in diastole. The nutrient arteries of the heart penetrate the muscle, generally, at right angles to the surface. They are thus clamped by the taut muscle in contraction, and the blood behind comes to a standstill. In diastole they suddenly open up and the blood goes forward under high pressure. This intermittent flow in the coronary arteries has been demonstrated in the heart

It has always been a puzzle as to why sclerosed coronaries should be so much more subject to thrombosis than are other arteries of similar size. I believe that in this intermittent flow we have a part of the explanation. With projecting banks of atheroma and shelving ledges of calcified deposit, favorable to the formation of whorls and eddies, increased by alternating flow and stasis, we have ideal conditions for thrombus formation. When the coronaries are once stiffened by sclerosis we may also well believe that alternate contraction of the heart muscle during systole, and stretching during diastole, may produce the vessel trauma that is said to be necessary for thrombus formation. In addition, the heart affected by infarct is almost always hypertrophied and dilated. This theory of trauma may find some support in the great frequency of thrombosis of the anterior descending branch of the left coronary which, in its long course, winds around the apex and is thus especially liable to stretching.

The blood flow in the coronary veins is continuous and rapid both in systole and diastole, and thrombosis in them is very rare. Dr. Waite has found one case in our postmortems. From the foregoing it will be seen that in the coronary circulation we have a very vulnerable part of the human machine. However, it repairs itself and keeps going in a most marvelous manner.

The exact cause of atherosclerosis remains unknown. But we are acquainted with some interesting facts. We know the composition of the atheromatous masses. We know that the process is not inflammatory, but one of a mechanical, chemical and metabolic nature. Just what initiates the process, we do not know. That the contents of these masses come from the blood stream, and that cholesterol is the transporting agent, seems to be established[2]. In the form of cholesterol esters the fatty acids are deposited in these lesions. They break down, setting free pure crystallized cholesterol and soaps. The al-

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kali of the soaps is later replaced by calcium and further by substitution of phosphoric acid; true bony deposit may occur.

Atheromatous changes are most common at points of stress and strain. Hence they occur about the conorany openings and also about the openings of the nutrient vessels of the heart muscle as they are given off from the coronary trunks. If one compares two opened coronaries, one healthy and the other atheromatous, he will be struck with the numerous vessel apertures in the healthy, as compared with the diseased vessel. The plugging of these small vessels by atheromata accounts for small patches of cariac fibrosis and infarcts too small to cause clinical symptoms.

It is said that in the early stages the atheromatous deposits may be absorbed and the vessel left but little changed. The atheromatous patches found on the valves in infancy are not seen in older children[2]. It was noted by German pathologists during the later years of the war, when fats were scarce, that atheromatous patches on the aorta were less common than in times of peace.

It has been demonstrated that the artificial atheromatosis of rabbits quickly disappears when they are again put on normal diet. These facts inspire the thought that perhaps even in advanced age some of the less sclerotic changes of atheromatosis may be amenable to treatment.

Arteriosclerosis has been produced in rabbits, guinea pigs and cats by diet, bacterial infection, drugs, and mechanical means. Diet has been the most successful. With the important work at the Santa Barbara clinic most of us are familiar[3]. It seems demonstrated that acid-producing foods are a factor in causing artificial arteriosclerosis in rabbits, at least, and clinical experience with Sansum's basic alkaline diet is certainly encouraging. Whether, as Newburgh, Sansum and Nuzum maintain, excessive cholesterol in the diet is not the main factor in atherosclerosis, as has been so positively asserted by certain Germans, is difficult to prove. Cholesterol[4] is apparently a normal constituent of the blood and its sources in nature are so numerous that it would be very difficult to exclude it by diet. Its antecedent is common in plants in the form of phytosterol, and cholesterol is common in many animal tissues.

Japanese experimenters have emphasized the importance of metabolism. Murata and Katoaka[5] found that feeding with lanolin produced more frequent and more marked atheroma in castrated animals than in normal controls. By raising metabolism in castrated animals with thyroid, the lanolin feeding resulted in little or no arteriosclerosis. Lowered metabolism as a factor in arteriosclerosis is interesting in connection with the opinion that lack of exercise plays a part in the human type. The addition of simple fats to cholesterol cause much more rapid and marked sclerosis than when cholesterol is used alone[7].

One fact stands out clearly in all these animal experiments, and this is that atherosclerosis in aimals may be produced by food,—by diet, and diet alone. The rabbit can hardly lay it on his grandfather, or civilization, or business worries, or other common alibi.

Now let us turn to clinical coronary sclerosis. In twenty-one cases verified at postmortem by the El Paso Clinical and Pathological Society, the average age was fifty-four years. None were under fifty years. The cases of cononary occlusion under fifty were all syphilitic. Syphilis is a common cause of coronary disease, but is not considered in this paper. Our cases were all males except one. Of Heberdeen's 100 cases of angina only three were females[8] In eighty-four cases of coronary sclerosis found postmortem at the Mayo Clinic[9], twenty-two per cent were females. Twenty-nine per cent of their cases were obese. All of our postmortem cases were of the robust, vigorous type, and many were decidedly fat. In fifty of my own cases of angina pectoris and cardiac infarct there were ten females. As to weight, nine were over 200 pounds, one weighed 265 pounds, one 250 pounds, three over 220 pounds. Seven weighed 180 to 200 pounds and six weighed 170 to 180 pounds. Thirty-two per cent were distinctly obese. Only eight weighed under 150 pounds.

In twenty-two cases the blood pressure was 150 or above, in many very high. In three it was below 110, all cases of cardiac infarct. My notes on past infections are not complete. None, however, had had an abdominal operation. This was also trùe of our postmortem cases, I believe. A history of acute inflammatory rheumatism was conspicuous by its infrequency, occurring in only two cases. I am doubtful as to the influence of focal infections. Certainly such foci are often not to be found. Nearly every case reported his previous health as exceptionally good. None were of the feeble, complaining, neurasthenic type. Angina pectoris is almost unknown among

those sufefring with pulmonary tuberculosis. We have found no case of marked coronary sclerosis in any case of tuberculosis at postmortem.

In reviewing the literature and my own experience, three facts appear prominent as to cause: (1) advanced age; (2) marked male predominance; (3) the vigorous overnourished type. Apparently, feeding experiments are quite as successful in humans as in animals. As to heredity I have no complete data. In high pressure cases it seems to be a factor. There is great danger, however, in confusing heredity with habits and environment. Habits certainly run in families, especially habits of eating, and I cannot help suspecting that what is too often inherited is an uncontrollable appetite. The older writers seemed to think so.

In 1842 Tweedie[10] observed: "Angina ordinarily occurs in persons of an advanced period of life, and more especially men who indulge in luxurious living and who, being exempt from the necessity of regular bodily labor, are prone to obesity; as well as from the striking benefit of depletion and reduced diet in a great proportion of cases."

In this country good living is not confined to the idle rich. The so-called laboring classes are able to enjoy angina and cardiac infarct too. I think the following quotation also throws light on the cause of coronary sclerosis. In a recent paper Joseph Pratt[11] said: "Doubtless coronary endarteritis and coronary sclerosis were very rare in Ireland or Stokes (Sir William, 1804-1878), one of the best clinical observers of his age, would have given us a vivid description of both angina pectoris and cardiac asthma. He says he never saw a classical case as described by Heberdeen and Latham."

Certainly Ireland had her share of tuberculosis and other infections. She certainly had her hardships and her worries. The well-fed Englishman had his beef and his suet pudding. The Irish had famine and potatoes. This, it seems to me, fits in well with the war experience of the Germans, and with the diet experiments on animals.

Primitive man lived by the chase and had periods of feasting and periods of fasting. The deposits of cholesterol and fat due to hyperalimentation had opportunity for removal. But today, in this country at least, the vascular system must bear the stress of three full meals daily, year in and year out. There is no time for repair. The coronary arteries become atheromatous, sclerosed and worn out; and angina, cardiac asthma, cardiosclerosis and cardiac infarct too often close the scene.

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THE SIZE OF THE HEART AS AN INDEX OF CARDIAC EFFICIENCY

ORVILLE HARRY BROWN, A. B., M. D., Ph. D.
PHOENIX, ARIZONA

Any factor which may assist in rating the efficiency of a heart is, of course, of value and should be heralded widely among the profession. Simple and fundamental as are the facts to be presented, I believe they are not being made use of as generally and fully as they should be. The manner in which a heart reacts to labor is the criterion of its ability to do that work.

The tests and the immediate response thereto of standing, hopping, running, of climbing stairs, etc., are of great value, but for some hearts any of these are too strenuous, while in other instances they might

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not easily suffice. Pulse rate and blood pressure changes may give some evidence of the efficiency of a heart; but many factors affect pulse rate and we know all too little as yet about factors concerned with blood pressure.

The question of efficiency of hearts is not simply one of life and death, but rather one of whether the heart is being called upon to do work for which it has been inadequately trained. Americans generally are heedless about putting undue stresses and strains upon their hearts. Office and professional men generally lead quiet, drab existences, as far as physical exercise is concerned, for months at a time, and then, with no preparation in physical training go into the mountains for a day, several days or longer, and hunt and do other strenuous things suddenly and thoughtlessly. All sorts of sudden physical exertions are undertaken even by persons with hearts subjected to toxemia from various sorts of focal infections. These persons, after having undergone undue physical exertion, may then return to their regular work with no period of marked rest. What has been the result on their hearts? By going back to facts early learned in physiology I hope to provide an answer.

The heart is a chambered muscle. All muscle has a property known as tonus. Tonus of muscle may be decreased; a stretched muscle—which is one with decreased tonus—is not an efficient muscle. Every medical student has suspended a weight on the end of a muscle with the other end attached to a lever writing on a kymograph. A fatigued muscle loses tonus, is lengthened and has not the ability to do work it had when fresh. A strip of heart muscle is no exception.

A structural phenomenon unlike that in any other organ of the body exists in the heart. The nutrient blood supply of the heart comes through vessels which lie upon its external surface. Whatever amount the heart muscle stretches, the coronary arteries stretch proportionately greater. A longitudinally stretched elastic tube loses width. The cross sectional areas decrease in ratio to the decrease of the squares of the diameters. I have shown in a previous publication that a heart might not be excessively dilated to, theoretically at least, have its nutrient blood supply cut down fifty or more per cent.

A heart may be put to sufficient work to cause it to dilate; and then, unless there is an unusual rest period, such a heart starts its regular routine, for example, on fifty per cent. of normal blood supply. Unless it has a great amount of reserve it may not be able to come back to its former efficiency, without a lapse of more or less time, or it may not come back at all without help. In many instances there is little chance of a heart's contracting to its normal size unless there is a diminution of its usual work or unless other therapy is instituted.

The grade of dilation of a heart may be anywhere from normal to extreme. The impairment of efficiency is somewhat in proportion to the degree of dilation. A heart may dilate so gradually that the stretch of the coronaries does not produce a corresponding narrowing of their diameters; or they may gradually widen so as to give a compensatory increase of their nutrient blood supply. In either event such a heart is not so efficient as it will be if it is again caused to contract and is kept at normal size. Its blood supply through the coronaries might then be greater than normal. The fact that so many dilated hearts are able to keep up a remarkable amount of work for long periods, indicates that some compensation must occur.

In the event that the coronaries are sclerosed a new factor enters. Atheroma is prone to be patchy. A vessel with sclerosed areas which cannot stretch, in the event of stretching must have other areas which stretch proportionately more. Therefore the narrowing at the stretched places is proportionately greater than if the entire vessel were stretched for an equal distance.

The long left coronary artery may help to explain why the left heart is found dilated so much more often than is the right heart. It must carry a proportionately greater amount of blood than does the right and hence, when dilated, the left heart has a poorer chance of recuperating and getting back to normal.

The etiology of cardiac dilation, as has been already indicated, is the lack of proper training of the cardiac muscle. Muscle has, in addition to the property of tonus, another property, that of developing more and more power as arise demands for more power. The one great physiological law in this connection is that the increased demands be made gradually and consistently. All muscle has a certain amount of reserve so that it can do work in excess of what it is actually trained to do. The heart which has been working for long in a dilated state has some of its reserve power used up. It is on this basis that a study of the size of the heart helps to decide upon the efficiency of that heart.

Toxic factors, such as focal infections,

tend to predispose to muscle weakness and hence to cardiac dilation. I wish to throw out a suggestion which, so far as I know, is new. The hint came from the experience with a patient who was heating her small apartment with a gas heater. She developed symptoms of cardiac embarrassment and I found her with a dilated heart. She attributed her condition to the gases from her stove. An electric stove was substituted for the gas heater and no further trouble of this sort was experienced by her.

I have been watching for dilated hearts, especially for eight to ten years, and I have sometimes feared I was finding them where they did not exist, so common have I found them. The thought has occurred to me that the carbon monoxide to which we are constantly subjected from our gasoline motors may be a contributing factor in the etiology of dilated hearts. At any rate the chronic effects of the carbon monoxide are worthy of more and serious study.

The symptoms of dilated hearts may vary from none to extreme dyspnea, cyanosis, edema and other signs of cardiac embarrassment. In cases with even moderate dilation there may be no symptoms of which the patient is conscious. After his attention is focused upon the symptoms, the patient may realize that he has had them for a considerable period. I have been struck with the frequency with which dilated hearts are found, even in those who give no histories that would point to the condition. Relief of the dilation brings relief, often, of the symptoms of which the patient has complained, which were, perhaps, only indirectly connected with the heart.

The diagnosis of cardiac dilation is not always easy. Locating the margins of certain hearts is indeed difficult. I make it a practice to utilize all the classical methods of examination—inspection, palpation, percussion and auscultation—checking the findings of one method by each of the others.

It would be presumptuous and trite for me to attempt to outline how these examinations should be made. It may be helpful, however, to suggest that inspection be made with the light reflected at various angles from the patient's cardiac area; that percussion be very, very light; that palpation be tried with the body leaning forward and with the chest in a state of extreme exhalation; and that auscultation be made on a plane with the apex beat and every cm. from near each axillary line or beyond, in, toward the heart, locating the point where there is the first sudden increase in heart tones. Examination should be with the patient standing or lying or sitting without leaning to either side. In difficult cases auscultation for the first change from dim to loud tones is more helpful, I believe, than any of the other methods. Fluoroscopic examinations are useful in checking the results from other examinations but, of course, can scarcely be made routinely.

Differential diagnosis is not usually important. Hypertrophy rarely confuses, because it rarely widens the diameter of the heart as much as one cm. This variation in the dimensions may be safely ignored. What is true of hypertrophy is also true of fat. Differential diagnosis between dilation and pericardial effusion may at times be a question; but the signs of effusion need not be detailed here. A heart may be retracted by adhesions or forced out of position in other ways so that both margins of the heart must always be located before its size is estimated. A vertical heart may be dilated and yet its left diameter be not without average normal measurements from the median line. The physician must base his judgment as to what are normal heart measurements for the individual upon indications as to the type of the individual. The size of the fist is a good basis by which to judge. The therapeutic test may be necessary to decide definitely that a heart is or is not dilated.

Prognosis as to the prospects of life and as to the prospects of usefulness may be given with some degree of accuracy. A person with a heart which remains dilated after careful treatment, or which readily relapses after the beneficial treatment, has not the same likelihood of work or life as the person with a heart which responds to treatment or which does not need treatment.

The treatment of a dilated heart is by regulation of exercise or the administration of digitalis or by both. The ideal method is absolute physical rest with speedy digitalization; digitalis is first decreased cautiously and then discontinued as soon as it is found that the heart remains normal size without it. Exercise should then be begun cautiously and in daily increasing amounts, insufficient to again dilate the heart. By this procedure the heart should be trained up to any required work. Failure may be due to a too rapid increase of work or to a lack of come-back, or reserve, in the heart. Practically, the treatment will often consist in the administration of digitalis and regulation of exercise. Very often such a plan of treatment will have to be protracted over months rather than weeks. All grades of responses to treatment, how-

ever, will be found. It is preferable, if possible, that a person continue regular work on a heart saving schedule, plus digitalis and supervision than that all work be stopped. The psychic factors should never be ignored.

The results of treatment are often most gratifying. Palpitation, arrhythmia, precardial pains, dyspnea, cyanosis, etc., are a few of the symptoms that are often benefited by proper attention to cardiac dilation. Other symptoms, not apparently connected with the circulation, are often improved by treating a dilated heart.

Occasional examinations after there has been a complete return to normal, should be the rule. The aged need more frequent examinations, as a rule, than do the youths. When the heart has been sufficiently improved, attention should be given to sources of toxemia. These should be eradicated but in a manner not to put further serious strain or stress upon the heart.

SUMMARY

Dilated hearts are frequently found if patients are carefully examined for them. A dilated heart is not as efficient as is that same heart when not dilated.

The chief cause of cardiac dilation is doing work for which the heart muscle has not been sufficiently trained.

Predisposing causes are toxemias. Focal infections are common sources of chronic toxemia.

DISCUSSION

(Papers of Drs. O. H. Brown and G. Werley.)

DR. W. WARNER WATKINS (Phoenix), opening:—If you are going to determine the efficeincy of the heart by its size, fluoroscopy is indispensable. I recall a paper by an eminent clinician of Tulane University, a year or two ago, in which he stated that not only could the size of the heart be determined within one centimeter of its transverse diameter by percussion, but that the heart could be distinguished from pericardial fat by the same means. I do not believe the ordinary clinician can do either of these things. It is a daily observation with us to check the heart size of patients whose percussion findings have indicated enlargement, and fluoroscopy shows normal size. Just a day or two ago a man was sent to our laboratory with request to determine the degree of enlargement. The patient was a large man with heavy chest, and the heart was almost infantile in size, much smaller than normal.

We do not believe it is reliable or accurate to use figures in estimating whether a heart is enlarged or not. The normal heart will vary widely in its transverse diameter, depending upon the size and shape of the chest cavity. We have seen an underweight patient, whose diaphragm was low and the heart somewhat pendant, show transverse diameter of not more than twelve centimeters; and three months later, when he had put on weight and his diaphragm had been pushed up, his heart was more transverse and measured 15 centimeters, and it was doubtless exactly the same size heart. When comparing figures, the relation of the heart to the thorax, the build of the patient, and other anatomical factors must be taken into consideration. We always supplement our figures by the statement that the heart is normal in size or abnormal in size for that particular patient. A transverse diameter which would be perfectly normal for a large man would mean a decidedly enlarged heart for a small man.

DR. S. D. SWOPE (Chihuahua, Mex.):—I would like to add a little matter of experience bearing on these two very excellent papers. In the first place, in the examination of over two thousand laborers in the last two years, belonging to our Mexican population, I have discovered the largest percentage of rejections for physical incapacity have been due to valvular lesions of the heart. The time allotted for this work prevents us from being able to make an exhaustive examination, and they are rejected rather summarily for this symptom.

A few months ago, I had the misfortune to be almost asphyxiated with carbon dioxide gas. My nurse and myself were attending an obstetrical case, and we discovered we were becoming lethagaric. The pains of the prospective mother had practically ceased, and it suddenly dawned on me we were being poisoned with carbon dioxide from an open charcoal burner in the room. The patient was moved and the pains commenced again. Our muscular incoordination was quite noticeable. In attempting to write, I found my muscles would not respond to the inclination of my brain at all, and I was practically unable to write my name, although I was not drunk, but I could not control the action of the muscles, and in walking I had the same sort of difficulty. This passed off and all hands recuperated after a short time.

I had a man who was completely asphyxiated with carbon monoxide. This man's heart beat for an hour and a half after this experience. He was probably in the smoke for fifteen or twenty minutes. There was no muscular action over his body anywhere, the heart was very slow, and there was no pulse discernible. We worked with him and gave him the ordinary artificial breathing, and the heart continued to beat for an hour and a half after we began working on him. The muscular action of the heart was very feeble, and continued to grow more feeble, until finally it stopped altogether. The respiration had stopped sometime before that.

DR. ORVILLE EGBERT (El Paso):—It is seldom that two papers on a program are so well correlated as the two last papers, as to give those of us so fortunate as to hear them such splendid instruction. It is as interesting a program as I have listened to.

From the standpoint of heart examination, following Dr. Watkins' discussion, I would like to hold up the end of the clinician a little. In percussing that heart and outlining it, I will agree with him that numerical measurements are inaccurate, because they may not take into account the relative size of the individual, and the measurements as outlined by percussion and the measurements as outlined by fluoroscope are both variable.

Someone at the University of Pennsylvania has developed a very complicated piece of electrical mechanism that I know nothing about, other than to say that in some sort of fashion, like in radio frequency, he has developed the electric impulse of the heart to where it is strong enough to trip his trigger and snap the electric plate, and he can, by changing his instrument, take the picture either in systole or in diastole, which does make for accuracy of measurement, but as the heart is in

action under the internist's percussing finger, I believe the relative measurements vary as much from the clinical standpoint as the other.

DR. O. H. BROWN (Closing): I wish to say just a word in appreciation of Dr. Werley's splendid paper. I have heard him several times, and it seems every time I have listened to the master speaking. He sent me a copy of the paper beforehand, which I appreciated very much indeed. He emphasizes the importance of diet, and I believe that as we grow older in the practice of medicine, we realize the importance of diet not only with the heart, but in other conditions. The objection is that there is so much poppy-cock about diet these days that this over-emphasizing it may run us into further danger. It is a question of learning facts and teaching them generally to patients and one another. I counsel against ignoring focal infections, even though you cannot see a definite connection between them.

The question of the fluoroscopic examination of the heart is, I think, extremely important. Dr. Watkins, I do not believe, would make us all x-ray men, but he would have us give some thought to the inaccuracies of our clinical measurements and examinations. Be as careful as we may, we cannot be correct constantly, but the proof of the pudding is the eating; take your patient and put him under proper treatment and see if you get response, and that can be found time and time again. I would, in that connection, emphasize what I tried to say originally, that this is a condition that happens so frequently that unless you have your fluoroscope handy all the time, patients will not be able to get out to the fluoroscope. You find dilated hearts many times with a very high blood pressure, and I have no fear of giving digitalis in high blood pressure.

I was glad to hear what Dr. Swope had to say, and I thank Dr. Egbert very kindly for his remarks.

As to the dropped heart, it is impossible to know just what the size of the heart of an ordinary man is, and if he has a dropped heart, it is more difficult to tell whether your measurements are varying from the normal. Valvular disease I have said nothing about. They affect secondarily the muscles that cause dilation. Myocarditis will very often disappear and allow the heart muscles to contract down to the normal.

DR. G. WERLEY (closing):—So much has been said, and all so well said, that I have very little to add. Dr. Brown had a fine paper, and his remarks were very much to the point. His paper will speak for itself.

A big heart is always a diseased heart, with very few exceptions.

I note Dr. Watkins' remarks about the fluoroscope, and his statement about the inability to measure the heart by physical means; however, I think we are about even on that. With the fluoroscope, if not careful and experienced, you will get a shadow that will throw you off as badly as percussion. Personally, I send more people for x-ray of the aorta than for the heart. You can also often tell by physical examination if you have sclerosis of the aorta. If the aortic arch can be felt, if the subclavian arteries are palpable and the aortic dullness wide, you certainly have aortitis of some kind. I think by using all your senses, you can usually get a pretty good idea whether the heart is big or not. I don't want to belittle the x-ray. We certainly need it, and need it badly sometimes, but what is important to us is what good is the heart; whether it is efficient or not. Just because you find a big heart, you are not through; you still have to determine what the efficiency of the heart is. If our patient can climb two flights of stairs without stopping, and without getting out of wind, he has a fair cardiac reserve and may be operated upon, so far as his heart is concerned.

I always look to see if the liver is enlarged, and listen at the back for rales, and look for dropsy and cyanosis. That will help you out a great deal.

Dr. Brown brought out the point of exercise. I would say, like Alfred Tennyson did, "there is no fool like the old fool," and when an old fool gets to trying to be young again, he is likely to meet disaster. Certainly it is very unwise for any man past forty-five, suddenly to conclude he needs to strengthen himself by going to a gymnasium class, or taking long hunting trips, or something like that, without gradually preparing himself. I do think a great many old people get into trouble with exercise. Walking is the best exercise for an old man, and when you get tired, stop—and maybe I had better stop.

INVESTIGATION OF DIARRHEAL DISEASES IN NEW MEXICO

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At the very outset I wish to emphasize the fact that the title of this paper should not be construed as an indication that the Public Health Service has come to any definite conclusions regarding the etiology of the seasonal diarrheal condition prevalent in New Mexico during the summer and early fall. We are not yet ready to say whether we are dealing with a new disease entity, an unusually large number of diarrheas with various causes, or an atypical manifestation of bacillary dysentery. Our tain results which indicate the line on which we believe further studies should be carried out, and it is with the view of stimulating the interest and cooperation of the physicians of the Southwest that the Surgeon General has asked me to appear before you.

For several years past the state health officer of New Mexico has been receiving investigations, however, have produced cerreports of an unusually large number of diarrheas among both resident and tran-

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sient population during the summer and early fall. These reports came from widely separated parts of the state, but, owing to the fact that they seemed to be more prevalent in the immediate vicinity of Santa Fe, it was decided to make that city the headquarters for our epidemiological survey. This survey was started about the middle of July of the present year and was discontinued on September 30th.

During this period, physicians from many different parts of the state of New Mexico have been interviewed and more or less intensive studies have been made in the following places: Army Rest Camp on the Mescalero Indian Reservation, in the southern part of the sttae, and Santa Fe, in the north central. At the latter point I, personally, saw many cases during their illness and tabulated the histories of seventy-two occurring in Santa Fe itself. In addition to these seventy-two histories, which were believed to be fairly reliable, twenty-three were collected from other parts of the state through the medium of the local physicians, bringing the total in which clinical data could be studied with some degree of accuracy, to ninety-five. In addition to these, we received blanket reports covering outbreaks at Las Cruces, Las Vegas and Hanover, New Mexico. These reports agreed in a general way with the findings at Santa Fe.

The onset was sudden in seventy-three instances; characterized by nausea and sometimes vomiting, in sixty-one of the ninety-five; abdominal pain in practically all (although this was usually of very slight severity) and a copious diarrhea in every instance except three, where constipation was complained of. The stools were watery in character and varied from two or three to twenty-four or twenty-five or even more in the first twenty-four hours. On the second day the symptoms were nearly always much better and very rarely were they severe after the third day. Watery stools were the rule, blood, as noted by the patient himself or his immediate attendant, occurring in but thirteen of the attacks, and mucus somewhat more frequently, having been noted by the patient or his attendants twenty times out of the ninety-five. All ended in recovery.

Slight fever sometimes occurred but it was such a minor symptom that the patient rarely noted it and there were not enough temperatures recorded in the series to be worth tabulating.

The disease was usually lacking in alarming symptoms and so short in duration that no physician was called—hence the difficulty of securing accurate histories or specimens of stools in large numbers. That the numbers were actually large is beyond question, because for every case in which we could get first hand information, we heard of many others who had been sick for a few days with similar symptoms but who had called no physician. Some of these we succeeded in locating and confirming the report, but many were among transients and tourists and no definite data was obtainable.

Although the symptoms were usually mild, none of the cases we studied having ended fatally, we occasionally received reports of deaths from gastro-enteritis in children and old persons in different parts of the state, with a symptomatology which suggested the possibility of an etiology identical with the milder type.

Out of the ninety-five attacks studied, there were seventy-seven first attacks, twelve second attacks, four third attacks, and two with more than three. Forty were males and fifty-five females. The average duration of the attacks was four and three-tenths days, with a median of three days and a modal distribution of four days. The average age of those attacked was twenty-seven and six-tenths years, the median thirty years and the modal distribution also thirty years. Out of the ninety-five attacks, the patients (or the mothers of the patients where the children were too young to express an opinion) attributed the attack to dietetic errors in only twenty instances, and, of these twenty, ten were children varying in age from one and one-fourth years to eleven years.

Besides going fully into the question of dietetic errors, inquiries were made in every case to develop any causal relationship between the attack of diarrhea and the following conditions:

1. Association with upper respiratory affections, including hay fever;
2. Contact with others affected;
3. Unusual sources of food;
4. The possibility of infection through milk or water;
5. Bites of insects;
6. Infected wounds;
7. Undue prevalence of house flies about the premises.

The result of this inquiry indicated that any association with upper respiratory affections and hay fever was accidental; that personal contact with others affected frequently occurred but that it was not an essential factor; that bites of insects and infected wounds were probably not involved, and that unusual sources of food or infect-

ed milk were not under suspicion. The one constant factor noted was undue prevalence of house flies, but whether this was a cause or coincidence, we are not yet able to say with any degree of assurance. Water infection did not seem probable except in one locality, the Zuni Indian Reservation, where an unusually severe form of diarrhea prevailed. Here only those persons, chiefly Indians, who drank the badly polluted river water came down with the disease. The white population, using deep well water, escaped.

The cases at the Zuni Reservation were in all probability true bacillary dysentery. They all gave a history of small, frequent, bloody and mucous stools, with fever and prostration, and frequently terminated fatally. This outbreak was plainly more severe and presented a markedly different clinical picture from the type prevailing in Santa Fe, the Army Rest Camp, Gallup, Las Vegas and other parts of the state.

Bacteriological studies of the stools were carried out by Associate Bacteriologist E. M. A. Enlows, who was sent from the Hygienic Laboratory for that purpose. She made cultures from seventy-four stools taken from fifty-six persons in our series of ninety-five. Among these fifty-six, she found nine different persons from whose stools she succeeded in isolating the Flexner type of dysentery bacillus one or more times. Besides these nine, she found the Shiga type in the stools from the only case we were able to study intensively on the Zuni Indian Reservation—an Indian child who was probably only one of many in that particular locality suffering from an attack of water-borne bacillary dysentery. The epidemiology of the outbreak at this place did not appear to have any unusual features and we refer to it at all only to emphasize the differences between it and the diarrheas we studied in other parts of the state.

One of the persons from whose stools the bacillus was isolated was a woman suffering from general ill health in which acute attacks of diarrhea was only an occasional factor. Another was a woman suffering from chronic infection of the antrum and having repeated attaacks of watery diarrhea of two to four days duration with complete recovery in the intervals. Neither one of these two cases gave any history of blood in the stools. There were two others in which the Flexner type was isolated, who gave histories of short acute attacks of watery diarrhea with no blood or mucus.

Besides this intensive bacteriological study on fifty-six cases of diarrhea, there was a control run by Doctor Enlows on one hundred and fifteen healthy children, varying in age from five to sixteen years. These were all negative.

The symptomatology of the nine positive cases of Flexner type was, on the whole, more characteristic of classical dysentery than the average of the series. Although the duration of five of them was only four days, thus conforming to the type studied, five of the nine also gave a history of blood in their stools, and a decided rise of temperature was noted in six.

Such, in brief, is the summary of our findings. Although the series is too small to justify definite conclusions, it is, we think, large enough to indicate the kind of questions we should ask and to weigh some of the pros and cons in favor of this, or that, answer. For instance:

1. Is the diarrhea we have described peculiar to New Mexico and adjacent states? We hope that the discussion will help us to answer this question. There must be many of you who have formerly practised in other regions of the United States, and, if so, we ask you to tell us whether you have seen widespread outbreaks of three to five day diarrheas, such as we have described.

2. Is it a new disease entity? Probably not—on general principles this is not likely. It has too many features in common with diarrheas of known causes. However, before this question can be answered definitely, many more stools must be subjected to intensive laboratory study and the clinical features we have described must be verified and checked up by other observers.

3. Is this disease, whatever it may be, water-borne? With the exception of the outbreak on the Zuni Indian Reservation, all the evidence we have is against such a theory. The water supplies, in so far as we studied them at other places, were not contaminated and the character of the outbreaks was not suggestive of water-borne infection. But this point also needs further study before giving a final answer.

4. Is it a form of food poisoning? We think not. The epidemiological data we have is against this view. However, there is a bare possibility that some widespread, universally-used article of diet may be infected in some hitherto undetermined way and the question of spoiled fruits, vegetables and milk, needs more study before final rejection.

5. Is it an aberrant form of typhoid or

paratyphoid? The epidemiological data does not favor this view and what laboratory evidence we have, is against it.

6. Is it a disease entity at all, or is it simply a symptom complex with a variety of causes? In other words it is sometimes bacillary dysentery, sometimes an acute indigestion caused by dietetic errors, sometimes an overwhelmnig dose of staphylococci or streptococci in the intestinal flora, or sometimes a combination of one or more of these causes? This is a long question and we think worthy of much more study before giving a final answer. In favor of such a view is the universal agreement that summertime, flies and camp life combine to produce intestinal disturbances of all known sorts and conditions. In those regions of New Mexico where we studied the outbreaks, we, of course, had the summertime, we ascertained that flies were abundant, and we also noted that we had, in effect, what amounted to camp life. In other words there were always many privies in the community unconnected with sewers and there were many new persons coming or going. In one mining camp there was a rapid labor turn over. In Santa Fe there were tourists, and at the Army Rest Camp, officers and their friends and families came from many different localities. In all the places where the condition was studied by us, we found abundant opportunity for a mutual exchange of intestinal flora from widely separated geographical regions, and before we give a final "No" to this question, we must ask and determine the answer to another, to-wit: Can one man's normal intestinal flora, non-pathogenic for him, be pathogenic for another, or, to put it from a slightly different angle, do we acquire an immunity to the peculiar strains of colon bacilli, streptococci and staphylococci of our own immediate neighbors and suffer an intolerance for those of strangers? So we answer this question with another and go on to our seventh query.

7. Is the disease we have been studying nothing but bacillary dysentery of the Flexner type? Against this view are certain features in the symptomatology as described by Wilburt C. Davison in his comprehensive monograph on bacillary dysentery, published by William Wilkins & Company, November, 1922, and which I beileve still represents the consensus of opinion of most internists and pediatricians. These features are as follows:

(1) General malaise, fever and headache, frequently present in the latter part of the incubation period of bacillary dysentery, were absent in practically all of our cases;

(2) Blood in the stools in sufficient quantity to be noted by the patient or his nurse, has always been considered as a **sine qua non** of the dysentery syndrome. In our series it was absent in most instances except in those in which the diagnosis was confirmed by the laboratory;

(3) The choleraform type, that is with vomiting and profuse, frequent and watery stools, is usually virulent, whereas in our cases these made as rapid and uneventful recovery as the others;

(4) Vomiting is relatively unusual, whereas in our attacks it occurred twenty-four out of ninety-five times;

(5) The pain at onset is usually severe —whereas in our series it was usually very slight.

Besides these clinical points of difference which seem to weigh against the theory of bacillary dysentery, is the outstanding fact that we succeeded in recovering the organism in only nine cases, where, theoretically at least, we should have recovered it in all we examined.

These, then, are the chief arguments which occur to us against the theory of bacillary dysentery. But, offsetting in a measure at least these contentions, are two other reasons of perhaps equal weight. The first is that the symptomatology of bacillary dysentery, especially of that caused by the Flexner type, is frequently atypical. For instance, a child in a family may have a typical attack of dysentery, being extremely sick, with small mucous and bloody stools, and running a course of ten days or longer, while other members of the family suffer at the same time with inconsequential watery diarrheas of only one or two days duration. Examples of this kind when the organism have been recovered from the stools of all the sufferers, mild and severe alike, are numerous enough in the literature to make us not decide offhand against the Flexner type of dysentery simply because the symptomatology does not subscribe to the classical picture.

The second reason which makes us give pause before accepting the arguments against this theory of causation is the undisputed fact that the organism of bacillary dysentery is extremely difficult to recover from the stools even in cases where it is known to be present. There are many reasons for this, all well attested, but which the limit of my time does not allow me to detail, and it may be that therein lies the whole explanation of why our search for the organism was only twenty per cent instead of 100 per cent successful.

SYMPOSIUM ON ACUTE APPENDICITIS

(Papers and discussions, based on the Clinical Records of three years, covering two hundred patients operated at St. Joseph's Hospital, Phoenix, Ariz., during 1924, 1925 and 1926. Presented at the January Staff Conference, January 10, 1927).

STATISTICAL REPORT ON 200 CASES OF ACUTE APPENDICITIS OPERATED IN ST. JOSEPH'S HOSPITAL, PHOENIX, DURING THE YEARS 1924, 1925 AND 1926.

W. WARNER WATKINS, M. D.,
Secretary of Staff

During the three years 1924-26, appendices were removed from approximately 600 patients in this hospital by 24 different operators. Of this number 200 were classed as acute appendicitis, and the study of this meeting is on this group, leaving the 400 cases of chronic appendicitis, including incidental appendectomies, for some future meeting.

Of the 200 cases diagnosed as acute appendicitis by the operating surgeon, there were fifty-two whose histologic picture was that of subacute or chronic changes, thirteen being classed as subacute and thirty-nine as chronic by the pathological examination. This does not necessarily mean that either the surgeon or pathologist was wrong, but this discrepancy of twenty-five per cent furnishes a serious problem from the standpoint of reliability of records. We believe it should be studied in some practical and impartial manner and steps taken to have the surgeons and the pathologist talk the same language.

Dr. Mills will discuss the pathological changes in the various stages of appendicitis. There are several cases in the record in which it is evident that the surgeon was basing his diagnosis on the acuteness and severity of the symptoms rather than on the changes in the appendix itself. When a surgeon makes a diagnosis of acute appendicitis, opens the abdomen and finds any visible abnormality in color, flexibility or feel of the appendix, he will almost invariably confirm his pre-operative diagnosis. From the standpoint of tissue change, if a patient has had symptoms more than four or five days, and his appendix has not become gangrenous or perforated, the inflammation will be subacute, because it is in the subsiding stage. On the other hand, cases diagnosed early, with every symptom of acute appendicitis, high blood count, nausea, vomiting, fever, pain and tenderness, and operated within forty-eight hours have been classed as subacute or chronic by the pathological examination. There is room for coordination of study. In ten of these fifty-two cases, which the pathologist classed as subacute or chronic, other possible causes for the symptoms are shown in the records. In these fifty-two cases the blood count was 12,000 or under in twenty-one cases, and over 12,000 in thirty-one.

In the 148 remaining cases, the blood counts were as follows:

Under 12,000	17
From 12,000 to 20,000	74
20,000 or over	50
No counts before oper.	7

The number of cases with white counts over 20,000 is rather surprising. The highest counts were in Cases 4731 (38,500, 90% polys), abscess which was drained; Case 5941 (37,000, 88% polys), abscess which was drained; Case 8385 (35,800, 88% polys), acute gangrenous appendix. The lowest count in the series was also a gangrenous appendix which had perforated (Case 6826), whose count was 4,800 with 94% polys.

The only statistical comment referring to etiology which we will make is to call attention to the preponderance of males over females, there being 117 males and 83 females. In chronic appendicitis, there is a striking reversal of these figures, there being 280 females to 120 males in that group of 400.

There were eighteen cases of acute appendicitis in children of 12 years or under, and here again the males predominated, there being eleven boys and seven girls in the list.

In the 200 cases, there were 55, or 22 per cent, in whom the appendix had ruptured or was gangrenous; in fifteen of these there was free pus or abscess formation.

There were fifteen deaths, or a post-operative mortality of 7.5 per cent. Brief summaries of some of these fatal cases follows:

Case 4692. M. 23. Patient had been sick a week, though had not seen a doctor, until day of operation. Had extreme tenderness and rigidity over entire abdomen. Appendix found ruptured with free pus; drain put in; appendix not searched for. Patient had albumen and hyaline and granular casts in urine; died on the fourth day, from peritonitis.

Case 5094. High School boy of 18. Taken sick two weeks ago; trouble subsided and went back to school. Six days ago temperature started up, with pain in lower abdomen; next day pain had increased, was nauseated; temp. 103, P. 90. Operation showed appendix abscess, Drained without removal of appendix. Patient apparently had septicemia and died after three days in hospital.

Case 5463. F. age 23, tuberculous but doing fairly well. Developed acute appendicitis and was operated promptly, but appendix was found gangrenous ruptured, with large amount of free pus in peritoneal cavity. Operated at 1:15 a. m., and died ten hours later.

Case 5629. M. 40. Has had tuberculosis for several years; has been sick a month before entering hospital; has palpable mass in right side. Abscess was drained without hunting for appendix. Fecal fistula formed and before death most of the bowel movements were coming through this opening. Lung and throat condition grew worse and patient died 30 days after entering hospital.

Case 6971. F. 44. Two weeks ago had severe abdominal pain in right pelvis and bladder; diagnosed peritonitis; this apparently localized in the right pelvis, and acute symptoms gradually subsided. At operation right tube ovary and appendix found bound together in mass which had multiple small abscesses. Following removal of this entire mass, peritonitis followed. Infection was streptococcic and patient died after several days.

Case 7370. M. 31. Taken ill July 8th, with pain, nausea and vomiting; castor oil and salts administered by physician on the 9th; on entering hospiatl on the 10th, there was extreme tenderness, rigidity and distention. Appendix was found gangrenous, perforated at junction with cecum; stump of appendix would not hold ligature, and appendix site was inverted. Died on second day after operation, of peritonitis.

Case 8086. Man. Had been having influenza; two days before was taken with pain in the abdomen, followed by nausea and pain referred to the gall-bladder region. Temp. was 104 and pulse 140 on entering hospital, with marked tenderness over entire abdomen. Acutely inflamed appendix, with abscess formed in the middle portion of appendix. There was a secondary drainage operation for peritonitis.

Case 8264. M. 39. Taken ill on Jan. 10th, with vomiting and pain; has had recurrent attacks of abdominal pain. Tender over the gall-bladder and around the side toward the kidney. Densely adherent retrocecal appendix, occluded at proximal end, bulbous at distal end. Developed a post-operative pneumonia and died.

Case 9474. M. 28. Three days before admission, developed severe pain over abdomen; grew constantly worse and his physician advised operation. Patient is tuberculous. Appendix found gangrenous, with involvement of bowel at base of appendix. Died three weeks after operation.

There was some discussion in the Advisory Committee about these cases, as to whether they were all to be classed as surgical deaths. After reviewing them carefully, it would seem that all of these fifteen cases are to be classed as post-operative mortalities. In some of them, tuberculous complications undoubtedly contributed to the fatal outcome, but none of them can be otherwise classed, with possibly one exception, the following:

Case 6500. M. 35. Patient with extensive tuberculosis of both lungs. For two weeks prior to admission has been having an attack, and formation of appendiceal abscess was diagnosed, but operation was refused. Appendix was found retrocecal and acutely inflamed, but no abscess. Fecal fistula formed and was repaired after a month. Patient had continued to have increased cough and dyspena, and finally died six weeks after first operation.

If this case, in which the patient apparently died from tuberculosis, is taken off, the mortality in all cases of acute appendicitis is 7 per cent.

This is not an excessive mortality, when compared with other statistics. The most recent report which I have been able to find is one by Warnshuis, who gathered the figures from seventeen hospitals in Michigan, Minnesota and Wisconsin; the mortality in some 5,700 cases was 4.35%; in those reports were hospitals with mortalities of nine, twelve, fifteen and one of 24 per cent. These, of course, were offset by reports showing very low mortality figures. Warnshuis's own personal report was of 327 cases with two deaths. When we consider the circumstances under which patients are referred for operation in Phoenix, many coming in by auto from outlying settlements, many in patients whose resistance is undermined by tuberculosis; many in Mexican people who have waited days before seeking medical advice; when we consider the very high percentage of gangrenous and ruptured appendices in this record, we can have little quarrel with a mortality of 7 per cent in 200 cases.

With regard to anesthesia, ether continues to be the anesthetic of choice with the majority of surgeons, with ethylene second and local anesthesia third. The following are the figures on the anesthetics used in these 200 operations:

Ether	120
Ethylene	30
Local	25
Nitrous Oxide	7
Gas-Oxygen-Ether	7
Local-Nitrous Oxide	5
Local-Ether, or Ethylene	6

HISTORY OF APPENDICITIS

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HISTORY

A knowledge of the morbid conditions affecting the vermiform appendix belongs exclusively to the nineteenth century.

The earliest definite anatomical record of disease in the appendix was reported by Lawrence Heister in 1775 (Med., Chir. and Anat. Cases and Observations).

The report reads as follows:

"In the month of November, 1711, as I was dissecting the body of a malefactor in the public theatre at Altodorff, I found the small guts very red and inflamed in several places, insomuch that the smallest vessels were as beautifully filled with blood as if they had been injected with red wax in the most skillful manner, after Ruysch's method. But when I was about to demonstrate the situa-

tion of the great guts I found the vermiform process of the caecum preternaturally black and adhering closer to the peritoneum than usual. As I was about to separate it by gentle pulling it asunder, the membrane of the process broke, notwithstanding the body was quite fresh, and discharged two or three teaspoonfuls of matter. This instance may stand as a proof of the possibility of inflammation arising and abscesses forming in the appendix as well as in other parts of the body, which I have not observed to be much noticed by other writers, and when in practice we meet with a burning and pain where this part is situated we ought to give attention to it. It is probable that this person might have had some pain in this part, but of this I could get no information."

The first reported case of appendicitis observed during life, is the classical one of Mestivier, in 1759 (Jour. de med. et chir. et phar., 1759). A man of forty-five sought relief for a tumor in the right side of the umbilical region. Fluctuation could be detected and about a pint of pus was evacuated by incision. The wound healed readily, but the patient died suddenly shortly after the operation, and at the autopsy the cecum was found covered with patches of gangrene, but otherwise presenting nothing abnormal. The vermiform appendix contained a large pin, very rusty, and so corroded in places that the least touch would have broken it. 'It is easy to understand," continues the report, "that, although the patient had never spoken of swallowing a pin, the one in question had been concealed for a long time in the vermiform appendix of the caecum, and that it was undoubtedly this which had irritated the different coats of which the organ is composed and given rise to all the patient's symptoms, finally causing the death which ensued."

In the year 1766, a medical student, Joubert Lamotte, published a report of a case in which the patient died with supposed intestinal obstruction, and at the autopsy the appendix and cecum were found filled with what were supposed to be cherry stones, and were evidently the first fecal concretions on record.

With the advent of the nineteenth century, the appendix began to be more frequently mentioned. In 1808 Jadelot reported the case of a boy who died of an "adynamic fever," where lumbricoid worms were found in the appendix at the autopsy.

In 1812, a London physician, Parkinson, published the case of a boy of five, who died of an attack of what was evidently acute appendicitis, and the autopsy showed a perforation of the appendix. This is the first case in which perforation of the appendix is recognized as the cause of death.

In the year 1824, there appeared an article by a Frenchman, Louyer-Villermay, which at once established a definite place for the lesions of the appendix in the category of recognized diseases (Arch. gen. de med., 1824). It contains the report of two cases in which both patients died after a brief illness characterized by violent pain in the right side of the abdomen, and vomiting. In both autopsies the appendix was found gangrenous.

During the first thirty years of the nineteenth century all interest in the vermiform appendix centered in France and England, but in the year 1830 a student of Puchelt, Goldbeck, published an inaugural dissertation on tumors in the right iliac fossa, which stimulated inquiry in Germany just as it began to decline in France, and for the next twenty-five years all important communications upon the subject proceeded, with few exceptions, from Germany.

During the period of time when Germany occupied such prominence in the literature of the appendix, four important contributions to it appeared in England—by Copland, by Hodgkin, by Bright and Addison, and also by Burne.

To Copland belongs the credit of first discriminating between inflammation of the appendix, inflammation of the cecum, and inflammation of the pericecal tissue.

The papers by Hodgkin and by Bright and Addison, contain excellent descriptions of the condition in question as observed upon the autopsy table, descriptions so clear and well presented that they could not be surpassed today.

Burne's papers somewhat hindered progress by the introduction of the term "typhso-enteritis." The unfortunate term "perityphlitis" invented by Goldbeck and his master Puchelt, had already diverted attention from the organ really at fault.

In the year 1837, the first American contribution to the history of the appendix appeared. Wolcott Richards, of Cincinnati (West. Journal of Med. and Phys. Sci., 1837) published a case in which the patient, a man of thirty-five, had a distinct chill followed by fever, but without pain or vomiting. On the fifteenth day he died in sudden collapse, and the autopsy showed a general peritonitis with recent adhesions, while the pelvis was filled with fecal matter issuing from a large ragged perforation in the appendix. Dr. Richards comments upon the fact that not only the vomiting but abdominal pain and tenderness were absent during the entire illness, and only after the final change for the worse was there extreme tenderness in the hypochondrium, with slight distention. In the next year a sim-

ilar case was reported in Philadelphia by Edward Hallowell (Am. Jour. Med. Sci., 1838) and these two cases, with four others, were all that appeared upon the subject in the United States for nearly twenty years.

The year 1838 was distinguished by the first operation for disease of the appendix as such. The aggressive surgery of the appendix as practised today, is a development of the last forty-five years, but the incision and evacuation of old encysted collections of pus in the right iliac fossa was practised as far back as the beginning of the Christian era. The treatment by incision and drainage at the point of fluctuation was practised and taught by Dupuytren, but the idea of incising the tumor before fluctuation appeared did not occur to him or his followers.

The first decisive step in the direction of modern methods was taken by a London physician, Hancock (Lancet, 1848) who, after making a diagnosis of inflammation of the appendix, incised the mass in the right iliac fossa without waiting for fluctuation to appear.

By the middle of the nineteenth century the true nature of disease of the appendix began to be recognized by the medical profession in general, as shown by a glance at the literature of the period. The obstacles which had blocked the progress of knowledge gradually disappeared, and after the year 1860 disease of the appendix became more and more a question of surgery.

In 1867, Willard Parker, of New York, published a report of four cases in which he treated abscess in the right iliac fossa, consequent upon inflammation of the appendix, by incision and evacuation. The first three cases, which occurred at intervals during the twenty years preceding Parker's report, were operated upon after fluctuation had appeared, but during this period Parker became convinced that it was not necessary, nor even desirable, to wait for fluctuation before making an incision, and the last of these four cases offered him an opportunity to put his theory to the test. The conclusions which Parker drew from this successful experiment were that nature attempted in such cases to protect life by building a wall of adhesions around the abscess but that ulceration of this protective wall might give exit to the contents. The question for the surgeon to consider, therefore, was, what could be done to assist nature in her efforts and at what period would his assistance be most useful. In Parker's opinion, the best time for the incision is between the fifth and the twelfth days, that is to say, after the adhesions are fully formed and before the maximum amount of pus is reached. The case in which Parker put this theory to the test was operated upon on the sixth day, when there was an area of circumscribed tenderness in the right iliac fossa, but no definite swelling, still less, fluctuation. An incision was made through the skin, beginning about an inch above the anterior superior spine and extending for about six inches toward the symphysis. As soon as the transversalis muscle was reached, the tumor could be felt, and it was freely opened, about four ounces of pus being evacuated. A tent was then inserted and the wound left to heal by granulation.

Parker's paper at once provoked discussion in many quarters, and the method he employed came into general use as the "Willard Parker operation." The medical profession had, by this time, acquired the anatomical and pathological knowledge necessary for many similar advances, and the only thing needed to inaugurate a new era in the treatment of the disease was a guarantee of safety in operation. During the year following the appearance of Parker's paper, this fundamental necessity was supplied in the principle of antisepsis, introduced into surgery by Sir Joseph Lister, and the surgical history of the appendix began.

It is also of interest to note here the reduction in mortality after the introduction of Parker's operation. In 1867, the death rate from inflammation of the appendix, under the non-operative treatment, was forty-seven per cent, while in 1882, when Parker's operation had been in use for fifteen years, it was reduced to fifteen per cent.

Up to this time, that is to say, the early eighties, incision before the detection of pus was the most daring procedure which surgeons had ventured to propose, but the time had now come when a much more radical measure was to be adopted and the actual removal of the appendix became the point upon which the eyes of the surgical world focused.

In 1884, Professor Mikulicz, of Krakow, performed an operation in which the appendix was ligated and removed in toto; this was published in 1886 and was the first instance of celiotomy followed by resection of the appendix, both as to date of performance and date of publication.

In the year 1886 an article appeared in America, by Reginald Fitz, of Boston (Am. Jour. Med. Sci., 1886) in which the word "appendicitis" appears for the first time.

The first operation on the appendix performed in the United States, was done by R. J. Hall, of New York, on May 8, 1886. The patient was a boy of seventeen who had had an irreducible hernia for several years. During the course of inflammation the hernia became strangulated; he was operated on for strangulated hernia, and the appendix was found.

The first successful laparotomy followed by removal of the appendix, undertaken with that possibility in view, was done by Thomas G. Morton, of Philadelphia, on April 27, 1887.

ETIOLOGY OF APPENDICITIS

The causes of appendicitis may be put into three divisions: (1) predisposing; (2) exciting; (3) final, or essential.

I. PREDISPOSING CAUSES may be local and general.

The appendix is a blind sac of relative great length and small caliber, and is very abundant in lymphoid tissue. It also borders on a cavity that is rich in bacteria.

Age. Appendicitis is a disease of early life. It is common between the ages of ten and thirty. A series of cases at the Johns Hopkins Hospital showed seventy-eight per cent under the age of thirty. The well recognized susceptibility of lymph tissue to infections during early life, is the probable explanation of this.

Sex. There is a greater liability in the male sex. The reason for this is probably because of the greater liability to exposure to injury and errors in diet.

Nationality: The negro race is relatively exempt from appendicitis. In the Johns Hopkins Hospital the relative admissions of whites to colored are 4 to 1, the cases of appendicitis are 12 to 1.

Hereditary Influence. Appendicitis frequently occurs in members of the same family.

II. EXCITING CAUSES:

Disorders of Digestion, such as constipation. In a series of cases at the Johns Hopkins Hospital 43 per cent had a history of constipation immediately preceding the attack. Sometimes the onset of the attack may be marked by diarrhea. An attack many times follows the ingestion of unsuitable food or a particularly hearty supper.

Menstruation. There is a relationship of the menstrual period and appendicitis. This is probably due to the congestion of the splanchnic area.

Trauma. This may be a direct factor in the cause of appendicitis in some cases.

Foreign Bodies and Concretions. A few years ago the origin of appendicitis was frequently attributed to foreign bodies such as the seeds of various fruits. Foreign bodies are rare, but at times are found, in the appendix. Many of the seeds described in the older literature were no doubt concretions. Intestinal parasites are sometimes found in the appendix, and may be a factor at times in the cause of appendicitis.

III. FINAL CAUSES:

The immediate cause of appendicitis is always microbic infection. The theory of stagnation and exalted bacterial virulence is no doubt true here.

The normal appendix contains in its canal the infective agent, which is innocuous to the healthy bowel. It is not necessary to introduce virulent bacteria into the appendix to produce inflammation, however. The aseptic ligature of the bowel forms a closed tube, and the consequent stagnation of the contents increases the virulence of the contained organisms. The tissue resistance is diminished and all the necessary conditions for an inflammatory outbreak are at hand.

Appendicitis may be a local expression of a general infection. It has been said that "Grip is the true cause of appendicitis." There may be an association of rheumatism with appendicitis, as well as tonsillitis. A relationship with various infectious diseases has been described.

SUMMARY:

1. A previous inflammation renders the appendix susceptible to further attacks.
2. The most important cause of appendicitis is digestive disturbance.
3. Acute or chronic enterocolitis may occasionally be an exciting cause.
4. Enteroliths and foreign bodies usually play a passive role. Pointed bodies and enterozoa may be direct exciting causes.
5. The clinical evidence is in favor of an intimate relation between appendicitis and rheumatic fever.
6. Animal experimentation and clinical demonstration make it plain that general infection is frequently the exciting cause of acute appendicitis.
7. It is not yet determined whether the general infection merely acts as an exciting factor by preparing a suitable soil for the activities of the intestinal bacteria, or whether the specific microorganism is the direct cause of inflammation of the appendix.

DISCUSSION

DR. S. D. LITTLE, Phoenix:—Doctor Greer has been so complete in his resume of the subject that I will just follow his outline and add a few remarks on the etiology of appendicitis.

In the **Relation of the Appendix to the Cecum** anatomically and physiologically there may be such a disposition of the mucous membrane at the point

of communication as to serve the purpose of a valve. There may be also a certain physiological spasticity of the circular fibers of the cecum at the point of communication with the appendix such as to form a temporary closure, locking up contents from the bowel for a time. These may be of such a degree as to produce or favor disease. This irregularity of the sphincter action has been noted particularly in x-ray examinations of the gastro intestinal tract. According to Doctor G. E. Pfahler it has a diagnostic value.

On account of the rudimentary structure of the appendix and on account of the want of any definite function, the appendix, about middle life or soon thereafter, undergoes an involutional change, so that finally it presents the appearance of a hard fibrous cord with scarcely any lumen. For this reason we find very little appendicitis after middle life. It has been suggested that the change may occur also as the result of an inflammatory reaction. The process usually begins at the tip and extends toward the base. Occasionally it is found localized some distance back of the tip while the tip is still apparently normal. This may be the result of a localized slow inflammatory process which may eventually lead to an acute exacerbation, producing the symptoms and signs of acute appendicitis.

Relation to Other Organs. The appendix usually has a mesentery, and at times this may be long enough to allow of a wide range of movement, especially of the distal end. When there is no mesentery, or when it is short, the appendix may be in close relation to the head of the cecum lying anteriorly, posteriorly, or to the outer or inner side. Any of these positions may be the determining cause of disease, but when the mesentery is long and the tip freely movable it may engage in an inflammatory process which is primarily either in the pelvis on either side, in the gall-bladder region, in the region of the stomach or pylorus, or in the opposite side of the abdomen. In some of these positions the appendix may become kinked in such a way as to favor disease.

Age and Sex. Young adults under thirty years, especially males, constitute the majority of cases. There are considerably more males operated on in acute appendicitis, while females are more numerous in the chronic cases. This disparity is supposed to be due to the fact that females are subject to more abdominal pains incident to menstruation and pregnancy and are more likely to defer operation.

Appendicitis is rare in children under two years, is claimed by most authorities. In one thousand cases reviewed by A. J. McCosh only four were under two years of age. However, W. B. Coley, of Columbia University, says: "Appendicitis occurs at all ages and is not uncommon at the first two years of life. It is more common in the first six months of the first year and the last six months of the second year. In eighty cases of appendicitis in infants, twenty cases were under three months of age, among which were two possible instances of prenatal appendicitis."

Constitutional Conditions are a distinct factor in the etiology of appendicitis. Out of 804 cases operated on by William B. Coley, constitutional debility was found in 57.7 per cent and a strong constitution in only 8.4 per cent. Familiar predisposition existed in 21.6 per cent of cases of destructive appendicitis and in 32.2 per cent of the catarrhal form. Poor nutrition was present in 49 per cent and good in 9.2 per cent. Nervous abnormalities existed in 76.8 per cent of the destructive, and 84.7 per cent of the catarrhal cases.

Doctor Greer stated that the immediate cause of appendicitis is microbic infection. Any microorganism, when favorable conditions are present in the appendix, may have pathogenic properties. Doctor John A. Lichty says the organisms usually found are the streptococcus pyogenes, the streptococcus hemolyticus, the bacillus coli communis, the influenza bacillus, and others.

Social Condition. The question as to the plan of entrance of the microorganism and the route it takes to the appendix, is not definitely answered. Some, among whom Aschoff is an exponent, hold that the infection comes directly from the intestinal canal; others, among whom Kretz is a leading exponent, insist that the microorganism comes from some certain focus, as the tonsils, and gains entrance to the appendix through the blood stream. Rosenow, in line with this latter idea, reports an extensive work in which he undertook to demonstrate that the microorganisms infecting the appendix have an elective affinity, and seek the tissue of this organ by special predilection. In this way he sees the close relation between diseased tonsils and a diseased appendix. Contrary to Rosenow's idea, Chastenet produced appendicitis in rabbits by feeding pathogenic bacteria.

Recently, while watching Doctor John Deaver operate, I heard him say that he has come to believe more and more that appendicitis, cholecystitis, and gastric ulcer are often the sequelae of focal infection. The fact that appendicitis may be caused by bacteria taken into the system through the alimentary canal or blood stream may account for the so-called epidemics of appendicitis reported in families, schools, and colleges.

Diet. The diet which most people eat seems to be a definite contributing cause. Most foods are prepared in such a way that mastication is scarcely necessary. Foods are taken hurriedly. The result is, that altogether too much work is thrown upon the stomach and bowels the cecum is compelled to receive the meal within six hours of its ingestion, no matter how poorly the digestive process has gone on. This may eventually lead to changes in the structures of the cecum, which are in turn taken on by the appendix, and disease occurs.

Trauma. As to trauma, a study of 1400 cases by J. B. Deaver showed that trauma was never the direct exciting cause of acute appendicitis in a perfectly normal appendix. However, trauma may promote trouble in an appendix already diseased.

PATHOLOGY OF ACUTE APPENDICITIS

H. P. MILLS, M. D., Phoenix, Ariz.

Appendicitis is an inflammation of the mucosa, muscular layers and interstitial structures of the appendix, frequently with extension to the peritoneal coat. Faulty drainage, due to narrow orifice, sharp kinking, fecaliths or, occasionally, foreign material, plays a part in its causation; unusual virulent bacteria in its lumen or a blood and lymph borne infection, represent the route of entrance of the infecting organisms. A large per cent of acute cases (some writers say practically all) show obstruction of lumen. This may be considered as one cause or as a result of the inflammatory change. Case in which the

proximal third showed no inflammatory change, and distal half was gangrenous, had obstruction at this point. The infection travels from the mucosa outward, the polynuclear cell infiltration spreading outward through the submucosa and muscular coats to the peritoneum in a very short period of time, within twenty-four hours—and often within five or six hours—all the layers being involved. Associated with this infiltration is found edema, hemorrhagic extravasation and necrosis, the latter, in the earlier stages, being in small focal areas and involving especially the mucosa. By the second day, or earlier, these microscopic necrotic lesions have coalesced and form macroscopic areas of necrosis leading the way to gross perforation of the appendix. The appendix is really perforated bacteriologically much earlier and involvement of the peritoneal coat occurs, so that the protection of the peritoneal cavity depends upon the development of, and maintainance of, integrity of the fibrinous protective exudate. Gross perforation, if it is to occur, usually takes place before the end of the second day.

By the third day, in the majority of cases, the changes have advanced to the maximum and attempt at repair begins. If gangrene or perforation has not occurred, microscopic evidence of repair is found in the presence of fibroblasts in the various layers of the appendix, especially evident in the subserous and submucous layers. In the gangrenous and perforative cases, the repair process proceeds more slowly, being first seen in vascularization and organization of the protective exudate, followed by liquefaction and absorption of dead cells and replacement by new fibrous tissue. Small ulcers of the mucosa are usually bridged over by epithelium, so that, in many instances, the patency of the lumen is preserved. The repair goes on quite rapidly so that, after a week or more, the microscopic picture is characteristic of a subacute or chronic process.

The blood findings in acute appendicitis are practically confined to changes in the number and relation of the leukocytes. The leukocyte count in a typical case shows a definite increase, with an elevation of the percentage of polynuclears. The total count varies from 12,000 to 20,000, occasionally higher. Such a count with less than 75 per cent polynuclears points to an infection of slight virulence or local resistance sufficient to limit its spread. A leukocytosis with polynuclear percentage of 85 to 90, or more, indicates an active advancing infection, probably peritoneal involvement. A high polynuclear count, in the absence of a leukocytosis points to a grave condition, the resistance to the infection being broken down.

In nineteen cases the post-operative diagnosis was chronic or subacute appendicitis, and pathological report was the same, with blood count of 12,000 or above. In all but four of these cases, the clinical findings and history, as given on the chart, point to other pathology which would account for the leukocytosis. In these four cases no explanation for the leukocytosis could be found in the patients' charts.

ACUTE APPENDICITIS IN ADULTS: CLINICAL FINDINGS

H. B. GUDGEL, M. D., Phoenix, Ariz.

SYMPTOMATOLOGY

The classical appendicitis characterized by slight fever, rigid right rectus muscle with greatest point of tenderness at McBurney's point, constipation and nausea, with early vomiting, builds up a set of clinical symptoms that make the diagnosis of appendicitis supposedly a mere trifle. But it so happens that many times the classical findings have missing links; and it is just that which makes interesting probably the most common of surgical diseases of the abdomen.

1. The Physical Signs of Acute Appendicitis. The patient is inclined to the right side, right thigh flexed. Facial expression, depending on the severity, shows suffering; usually pale. No great early changes in the contour of the abdomen; however, if peritonitis has developed, the abdomen is distended. Pulse, at the beginning, is wiry; in the later stages it shows variation, is weak, dicrotic and rather easily compressible. Tenderness may be observed over the abdomen but in most cases, after first few hours, centers at McBurney's point. Rigidity, while not an infallible sign, must be considered of much significance. The two recti are easily comparable. A circumscribed induration in most acute cases manifests itself after twenty-four or thirty-six hours; as the case continues the swelling more and more obliterates the natural curve of the iliac fossae. In those cases where the appendix is directed backward and upward (Case 5734) the tenderness appears to be just above the crest of the ilium, with a tendency upward in the axillary line. With the stethoscope there can be noted, many times, the fine rubbing like rales over the right iliac fossae.

2. The chief symptom, or the attractive symptom, of appendicitis is pain—abdom-

inal pain. This pain is at first colicky in character and usually, the first twelve hours, is more or less diffuse over the entire abdomen. As the condition proceeds, the pain gradually has its point of intensity in the right lower abdominal quadrant. Occasionally the right side is the sole location of pain and very frequently the patient complains of pain in right thigh and genitals (Case 4991). The final location of the pain is at, or near, McBurney's point. The cessation of abdominal pain, with continued symptoms, in appendicitis usually means rupture.

3. The point of extreme tenderness, as stated, is located at McBurney's point, which is described as at, or near, the middle of a line from the umbilicus to the anterior superior spine. According to MacKenzie, it is directly over small filaments of the last two dorsal nerves where they pierce the rectus. While McBurney's point of tenderness is usual finding, yet adhesions (as noted in case 9579, where appendix was attached to lower pole of the kidney; and again in case 9876, with adherence of appendix to a tubo-ovarian cyst) may make McBurney's point valueless. This is also true in Case 8908, where there were anatomical irregularities (a noted failure in rotation of the colon) completely changing the usual landmarks. Again, on account of adhesions to the posterior wall (case 5734 and case 7144).

4. Rigidity. The rectus usually shows this defense in protection (cases 6500 and 6324); while case 7154, acute appendix, states that no rigidity was found, proving that not always is rigidity necessary.

5. Constipation is the rule. I have never seen an uncomplicated appendicitis otherwise and no case report that I have examined has given diarrhea in the history. However, some authorities make the statement that diarrhea, associated with the usual symptoms, is a dependable symptom.

6. Vomiting rarely persists. While reflex, yet the retching and straining in emesis does aggravate or, at least, rather intensifies the abdominal pain.

7. Fever, while rather a constant early symptom, is occasionally absent, as noted in cases 6718 and 6197. Higher temperature, 104 or even higher, occurs. Fever cannot be considered a guide to the severity of the case; many case histories show severity with low temperature. Pulse is 90 to 120.

DIAGNOSIS

Ruptured peptic ulcer, pancreatitis, renal colic and intestinal obstruction, until infected, rarely produce fever or increase white count, though they show sudden nausea, vomiting, and pain.

Movable kidney (in Dietl's crisis) may give appendiceal symptoms, but the history of previous attacks and the excessive output of urine are distinguishing features.

Acute hemorrhagic pancreatitis will give just cause to think of appendicitis and peritonitis. The deep pain, shock, with a show of blood sugar, will assist in differentiating.

In acute tuberculous peritonitis, the onset is more gradual, although the pain, tenderness and fever, are similar to those symptoms in appendicitis. However, the right side resistance is not present and a more general abdominal dullness can be demonstrated.

Pyosalpinx gives history of infection, with presence of mass on vaginal examination. Case 9112 shows adhesion of appendix to right tube. Case 9071 shows an acute appendix with a right and left hydrosalpinx with twist in each pedicle. Case 5755 shows same, with intestinal fibroid, multiple ovarian cysts, and myofibroma.

Ectopic gestation gives history of suppressed menses, shows sudden onset, shock, pallor, no fever, no marked change in blood count.

Volvulus is usually located at the sigmoid. However, twist or adhesion may simulate appendicitis. Failure of bowel action, and later stercoraceous vomiting will differentiate.

Cholecystitis, with retention, in absence of jaundice or the pear-like tumor, is confusing, should the appendix on account of abnormal position be attached at or near the liver as noted in cases 6478 and 5932.

Perinephritic abscess (which was diagnosed by Dr. Murphy on all but his friends, by a punch in the back) may simulate appendicitis. The x-ray or exploration may demonstrate the difference.

Lobar pnuemonia, involving the right base, or pleurisy involving diaphragm on right, while not frequent in adults, is confusing when it does occur. The chest findings and rusty sputum will aid.

Lead poisoning gives history, blue line on gums, and lack of right rigidity.

Syphilis is differentiated by the abdominal lightening pain or Devil's grip. history, and blood findings.

In conclusion, I wish to compliment the physicians for, in the most part, rather complete histories, with special mention of author of history 9579-8908. It has been a real pleasure to try to review the 200 or more histories.

DISCUSSION

By DR. ORVILLE HARRY BROWN. Dr. Gudgel has given such an excellent and thorough discussion of the symptoms and diagnosis of acute appendicitis that it is with difficulty I have found anything at all to say without a reiteration of truths. I shall, however, attempt to avoid repetition.

One of the important points in the diagnosis of acute appendicitis is to get the patient and his family to accept the diagnosis in all its seriousness. I have nothing special to say on this subject except, "Do not stretch the truth; at least do not state an untruth in order to convince the patient or his family of the correctness of your diagnosis." Patients sometimes get well when it seems an impossibility.

The diagnosis of an acute case of appendicitis is, ordinarily, not difficult. It is sufficient, as a rule, to make the diagnosis of surgical abdomen. One of the possible errors is to diagnose a kidney or ureteral stone as an acute appendicitis. This would probably necessitate turning the patient over for another incision. It might be possible to mistake gallstones or a perforated peptic ulcer or other abdominal disease, for appendicitis. In such an event it ordinarily means only extending the incision a short distance and, possibly, adjusting the operating table.

If you diagnose every acute condition of the abdomen as acute appendicitis you will be wrong in less than 30 per cent of the cases.

Lichty says, in 100 cases of acute surgical conditions of the abdomen, 70.3 per cent will be appendicitis; 18.1, intestinal obstruction; 7.2, perforations; 2.4, cholecystitis; and 1.1 per cent, twisted pedicles.

As said by Dr. Gudgel, the outstanding symptom of acute appendicitis is pain, which, at first, may be referred to any place from the chest to the lower part of the pelvis. Its localization in the lower right abdomen usually results within thirty-six to forty-eight hours.

As I think back over my cases of acute appendicitis it seems to me that vomiting was relatively rare. Lichty says, however, of eighty-three cases, nausea occurred in seventy-two and vomiting in fifty-six of them. Seven of these eighty-three had diarrhea instead of constipation.

Dr. Gudgel, I believe, did not mention the importance of a rectal examination in the study of an acute case of appendicitis. This should rarely be omitted.

Another point worthy of mention is the pain that is engendered by a release of pressure over McBurney's area. If pressure is made gently and firmly over the appendix and the pressure is suddenly released, definite increase in pain in the area is noticed.

Livingston has called attention to the hyperesthesia found in the right lower quadrant of the abdomen as tested by pinching the skin between the fingers. Definite hypersensitiveness is found in the right lower quadrant. Another way of testing this hypersensitivity is by means of pin pricks.

I would reiterate one point made by Dr. Gudgel, which is, that muscle spasm is the most important diagnostic sign. It is an almost constant finding but it may have to be tested for delicately.

Differential diagnosis must consider anaphylactic reactions in the region of the cecum. Osler was the first to call attention to angioneurotic edema of the abdomen. Duke and others have proven the possibility of anaphylaxis giving symptoms resembling those of appendicitis.

Another condition which I have found several times is an appendix adherent about the end of the cecum, which, when the cecum is markedly distended, is stretched so that it is acutely painful. Therefore an acute appendicitis has to be differentiated from chronic appendicitis.

THE SYMPTOMS AND DIAGNOSIS OF ACUTE APPENDICITIS IN CHILDREN

D. Fournier, M. D. C. M., Phoenix, Ariz.

In childhood the order of appearance of symptoms is usually the same as in the adult. There is, first, generalized abdominal distress, commonly beginning with severe or sudden epigastric pain; second, within a few hours, nausea and vomiting occur; third, rigidity and tenderness appear in the right lower quadrant; fourth, elevation of temperature occurs within the course of several hours; lastly, leukocytosis appears.

Acute appendicitis begins with a pain of a sharp or colicky character in the epigastric region, usually sufficient to bring the child to its mother. Not infrequently the pain begins in the right lower quadrant. There is not the sudden, overwhelming, staggering pain of a perforation, but it is rather on the order of the old time "stomach ache," growing worse in the course of an hour or two. The sharpness of the pain may diminish but, unless spontaneous recovery occurs, it does not disappear.

A series of twenty-five children who came to this hospital for operation and whose histories were analyzed, offers some very interesting and instructive facts as to the character of the pain at the outset. Generalized abdominal pain of a dull or colicky character was present in twenty-one cases, or 84 per cent; and was absent in three, or 12 per cent; and in one case no history was given.

The character of the pain was colicky in ten, or 40 per cent; dull in two, or 8 per cent; dull and colicky in three, or 12 per cent; and unknown in ten cases or 40 per cent.

Symptoms in order of development: (1) generalized pain, (2) nausea and vomiting, (3) localization in the right lower quadrant. We may conclude that in the great majority of children the first symptom to usher in an attack of appendicitis is generalized abdominal pain, usually colicky in nature. However, generalized pain may be entirely absent, as was the case in 12 per cent of this series.

Localization of pain in the right lower quadrant was present in eighteen cases, or 72 per cent, and absent in six, or 24 per cent.

In the order of appearance of symptoms,

pain in the right lower abdominal quadrant was the first symptom in eleven cases, second symptom in seven, and in six no record was made as to the order of appearance of symptoms.

The nausea which early follows the onset, commonly terminates in vomiting and the vomiting may be repeated once, twice or oftener during the day. While emesis is very frequent in any of the affections of childhood, still, nausea alone may be present. Vomiting is very constant but not persistent during the earlier stages of the disease. In the later stages of the disease, following rupture of the appendix, vomiting may reappear as a symptom of peritonitis.

Of the twenty-five cases studied, vomiting was present in fifteen, or 60 per cent, at the onset, and in nine cases, or 36 per cent, it was absent. Nausea alone occurred in one case.

In the course of several hours following th onset of appendicitis, the temperature begins to rise. The temperature may range from 99 to 103 degrees F.; while a temperature over 103 degrees is rather suggestive of trouble elsewhere.

Pulse and respiration usually correspond to the temperature. When there is a spreading peritonitis the pulse becomes very rapid, weak and often irregular.

Chills at the onset of appendicitis are rare. Constipation is very frequent in these cases and is usually well marked, and it may reach a degree suggestive of a mechanical obstruction. Diarrhea occasionally ushers in an attack. The practice of the laity of administering cathartics often masks the true state of affairs, and is to be condemned.

The examination of the abdomen in a child old enough to express its feelings, is as fruitful of results as in an adult. It should be carried out, however, with much tact. The child's confidence should be gained. Rough handling should be avoided, as the fear it excites at once eliminates any value one might get in palpation as an aid to diagnosis. The child should be in bed or on a pillowed table. He should lie with head, shoulders and knees well raised and supported. The examiner's hands, as well as his heart, should be warm. A warm heart will gain the child's confidence.

Two points are made out on palpation: a localized area sensitive to pressure, and an area of localized involuntary rigidity. In palpating for an inflamed appendix, one of the most constant findings is an involuntary muscular reaction which is known as rigidity. Abdominal distention is usually an indication of extension to the peritoneum, and is a late development. The child is frequently found lying on its right side or back, with the right thigh flexed on the abdomen.

The symptoms of appendicitis in infants are similar to those in older children, but there are some points of difference, as a rule, in infancy. The disease has a more insidious onset, and it has a tendency to develop rapidly into a general peritonitis, as happened in one of the cases studied in this hospital, in an infant one month old. This was a very interesting case; first, on account of the age of the child, and secondly, in the multiple post-mortem findings. The result of the post-mortem showed the child had a lobar pneumonia, a general peritonitis, an acute hepatitis, a congenital stricture of the urethra, acute nephritis and an early acute appendicitis. This child did not have as much chance as the proverbial snowball.

In making a diagnosis of appendicitis, the main points to be considered are: (1) history of a previous attack; (2) situation of pain; (3) rigidity of lower portion of right rectus muscle.

Of all the sources of difficulties in the diagnosis of appendicitis in children, gastro-intestinal disturbances easily head the list. Differential diagnosis should be based on the history, the symptoms and signs, and a complete examination of the entire patient. In making a differential diagnosis we have to consider those conditions outside the abdominal cavity, namely, infections of the respiratory tract, the exanthema, and typhoid fever. In differentiating it from pneumonia, a few points may be emphasized. Pneumonia gives a previous history of cough and cold; the abdominal pain is severe and constant, while that of appendicitis is paroxymal, less intense; vomiting is much more prevalent in appendicitis than in pneumonia. Abdominal tenderness is more marked on deep pressure in appendicitis than in pneumonia. The temperature in pneumonia ranges from 103 to 105 degrees, while in appendicitis it ranges from 98.6 to 102 degrees.

In acute intestinal obstruction, the onset is more abrupt and the pain is more severe. Tympanitis develop early and vomiting is very persistent. There is an absolute constipation.

Intussusception is characterized by intermittent pain, colic, vomiting which is severe from the outset, and bloody stools. Acute nasopharyngeal infections may at times be very confusing. Urinary tract infections are common and a fertile source

of error. Pyelitis on the right may be difficult to differentiate from an appendicitis. It is more common in girls. The temperature is rather high and the urinary changes are marked.

CONCLUSIONS

Every acute abdominal condition in children should be looked upon as appendicitis until it is proved otherwise.

The dangers and frequency of appendicitis in children should be explained to parents. The wholesale giving of cathartics to children in every abdominal complaint should be warned against, as it is often a dangerous procedure. Parents should be instructed in this danger.

Discussion by DR. JOHN WIX THOMAS:—This is such a comprehensive paper that there is little to be added to it, and only one or two points which will bear emphasis. It is very advisable to teach people to avoid cathartics in sickness that comes in the home, especially among children. Another point is that rigidity of the rectus muscle is one of the most constant symptoms and is present in the majority of cases. None of the symptoms are present in all cases. It is a mistake to rely on any one symptom, but must look at the picture as a whole. Another point is the leukocytosis. This does not appear early in every case, but is one of the principal points for differentiation.

TREATMENT OF ACUTE APPENDICITIS

E. PAYNE PALMER, M. D. F. A. C. S.
Phoenix, Ariz.

Acute appendicitis is a surgical disease amenable only to operative treatment. The patient should be operated upon as soon as a definite diagnosis is made and the patient can be taken to the hospital and prepared for operation, unless there is some contraindication. Statistics of insurance companies show that there are practically no deaths in appendicitis operated upon before the end of the second day of an acute attack. If, for any reason, operation cannot be performed, the Ochsner treatment (rest and starvation), if properly applied, will save a number of lives.

Appendectomy, during an acute appendicitis, may be a very simple procedure but is frequently one of the most varied and difficult of operations. The McBurney incision is not satisfactory for exploration and, in adults, a considerable percentage of such incisions are followed by inguinal hernia within from one to two years after the operation, because of muscular relaxation at the inguinal ring, probably due to the injury of a nerve.

A straight, low, right rectus incision is greatly to be preferred as it permits of exploration and can be enlarged if needed. I prefer the Battle incision when the case is early and not expected to be difficult, but, in the very acute cases, a low, right, paramedian incision which is long enough to give a free exposure of the abdominal cavity in order to be sure that no infected areas are overlooked, to permit of sufficient protection of the remainder of the peritoneal space, and to allow free handling of the appendix without danger of damaging other structure. In the average case, a three or four inch incision will suffice and, if necessary, this can be enlarged. Extreme care and gentleness should be used in making out the position and relation of the appendix. When this is done, the field of operation should be isolated by the free use of moist gauze abdominal packs until there is no danger of infectious material being spilled into the abdominal cavity, and only the inflamed appendix and the parts immediately adjacent to it, remain exposed. Frequently, the inflamed appendix is buried in a mass of dense adhesions, and again extreme gentleness must be used in finding a line of cleavage so the appendix can be isolated and delivered. This is frequently extremely difficult and even the most expert operator may have difficulty in distinguishing between a greatly distended appendix and a small intestine. When the appendix is delivered, a gauze strip should be pushed down into the bed from which it came.

There are essentially two methods of doing an appendectomy. The inversion, or burying, of the stump and the ligation and drop method, leaving the stump free. I prefer and practice the former, although the latter has many claims of superiority made for it. If a perforation, or gangrenous area which is in danger of perforating, is found, the base of the appendix should be crushed with a clamp, a catgut ligature tied into the groove and the appendix cut away at once, to prevent spilling of infectious material. Rarely, the cecum at the base of the appendix is gangrenous and must be closed with double layers of catgut sutures. In these cases it is good practice to stitch the omentum over the wound.

The field of operation should be dried and the gauze packs removed and the omentum brought down into the field of operation. If the case demands drainage, a large split rubber tube is passed down to the cecum and another into the pelvis, and the abdominal wound closed. The tubes are usually removed in from three to five days.

The large appendix abscess should be dealt with as an abscess elsewhere would be—open into the most accessible part and

establish free drainage, from the lowest point if possible. The removal of the appendix in this class of cases has been hotly debated. If the appendix is found after the simplest examination, remove it; otherwise, leave it. Recurrences of appendicitis after an abscess of the appendix has been opened and drained, occur in about ten per cent of the cases. It is, therefore, advisable to remove the appendix in these cases, to avoid recurrence of the attack, after the patient has thoroughly recovered from the appendix abscess and there is no more drainage of pus.

The postoperative care is most important and the success of the operation frequently depends upon it. Fowler position is used in the majority of drainage cases. Proctoclysis is given almost invariably for twenty-four or forty-eight hours, or until water can be taken by mouth freely. Glucose, alone or with soda, is added to the proctoclysis if it is deemed advisable. Hot bicarbonate soda solution is given by mouth as soon as the patient asks for a drink; when there is no nausea or vomiting, tap water is given in increasing quantity. Morphine should be given in sufficient quantity to make the patient comfortable. It should never be withheld if the patient is suffering. Cathartics should never be given; many deaths have been attributed to purgation. In their place, use from two to four ounces of olive, or cottonseed, oil as an enema, on the evening of the second or third day, to be retained over night if possible without discomfort. The following morning, use from one-half to one pint tap water as an injection. This procedure can be repeated daily until the bowels act normally. Liquid nourishment is begun on the third to fifth day and soft food is gradually added; the giving of solid food is usually delayed for from seven to ten days. When the patient's condition will permit, the sooner he is out of bed the better. The non-drainage cases usually get out of bed in five to seven days and may return to light work in from two to three weeks.

These life-saving details have been carried on in fifty-eight cases of acute appendicitis which we have operated upon in this hospital during the past three years: these have failed us four times. The cases operated upon have covered every phase of acute appendicitis from the mildest attack to large abscess. The leukocyte counts have ranged from 4,800 to 37,800. We have learned that the subjective symptoms and the physical and blood findings, must be correlated in order to arrive at a diagnosis.

The following four cases are interesting from the standpoint of the leukocyte count and illustrate the importance of early diagnosis and early operation. Early operation is not only a life-saving procedure but, from an economic standpoint, is of great importance to the vast majority of patients. If operated upon early, complications are avoided and the patient is restored to health quickly and is saved the expense of long hospitalization.

Case 5049, Mr. J. E. G., aged 41, realtor, was admitted November 15, 1924. At 2 p. m. the patient was awakened with pain in the abdomen, which increased severity. Pain was first general but localized in the right iliac region. There was a marked tenderness and rigidity over the right iliac region. The patient was seen five hours after onset of pain. Leukocyte count 29,900; polynuclears 94 per cent. Operation was performed one hour later by inversion method, which revealed a much enlarged edematous appendix. Abdomen was closed without drainage. Patient was up on the sixth day; left the hospital on the eighth day, cured.

Pathological findings: Section showed diffuse polynuclear infiltration area with necrosis.

Case 6826, Mr. H. W., aged 56, cook, was admitted March 18, 1925. Twenty-four hours previous to admission, patient was seized with pain in the lower right quadrant of the abdomen, became nauseated and vomited. The pain was generalized at first but, after three hours, localized in the lower right iliac region. Had no medical attendant until shortly before he was admitted to hospital. Examination revealed a marked rigidity and tenderness over the entire abdomen, most marked over the right iliac region. Temperature was 100; pulse, 120; the leukocyte count was 4,800, with polynuclears 94 per cent. Operation revealed a ruptured gangrenous appendix with large amount of fluid in abdomen. Appendectomy by inversion method was performed. Abdomen was closed with drainage. Patient made a most satisfactory recovery and left the hospital April 6, 1926, cured.

Pathological findings: Section showed diffuse inflammatory infiltration and necrosis.

Case 8385, Mr. E. S. Y., aged 47, rancher, was admitted January 1, 1926. Patient dates trouble back about two months, when he had a severe pain in the right side. Physician made a diagnosis of appendicitis and advised operation, which was refused. Patient was in bed three days during this illness. Two days previous to entering the hospital, patient had severe pain in the right side and his physician advised him to enter the hospital for immediate surgical treatment. This advice was not taken and it was not until the pain was excruciating that he consented to operation. Examination of the abdomen showed it to be markedly distended, with generalized muscle rigidity and tenderness most marked over the right iliac region. Diagnosis of acute appendicitis with probable rupture was made. The leukocyte count was 35,800 with polynuclears 88 per cent. Operation revealed a large gangrenous appendix filled with pus and a large amount of exudate on the intestine adjacent to the appendix. The ligation and drop method, leaving the stump free, was used, as the head of the cecum was too edematous for purse string suture. Abdomen was closed with drainage. Patient made an exceedingly satisfactory convalescence and left hospital February 4, 1926.

Pathological findings: Appendix covered with plastic exudate. Section showed diffuse polynuclear infiltration and there was extensive necrosis.

Case 5941, Mr. E. A., aged 20, was admitted August 20, 1924.

History: No hereditary diseases in family history; mother's health is fair; father deceased, cause unknown; patient had four brothers and two sisters, all in good health. Patient has had all usual diseases of childhood, made a good recovery; tonsillectomy three years ago; and has pulmonary tuberculosis. Patient suffered severe pain for four days in the lower portion of right abdomen, with vomiting and constipation. Pain became much worse and, on the date of admission, he was unable to arise from bed.

Chest findings: Anterior and posterior vocal fremitus; prolonged expiration of upper and middle lobes; few rales at apex both anterior and posterior; plastic pleurisy at base extending around right lung to left chest, anterior. Increased vocal fremitus; prolonged expiration; delayed resonance from fourth rib to apex; rales over the entire lung from base to apex; posterior much the same condition, rales over entire lung.

Examination showed marked rigidity over entire abdomen, tenderness over the lower portion of the right iliac region, with a definite fixed mass. Incision over the center of the mass revealed a large abscess from which a concretion was removed. Appendix was not sought for. Large drainage tubes were inserted. Patient had a stormy convalescence but made a satisfactory recovery and left hospital November 24, 1924.

DISCUSSION

DR. A. M. TUTHILL:—Acute Appendicitis is like any other acute operation, the main point being to get in and get the appendix out with as little harm as possible. There is one point on which no one has laid any particular stress and that is the triangle of hypersensitiveness in acute appendicitis; this runs from the umbilicus to the crest of the ilium, from there to the right pubic spine and back to the umbilicus. Elicit the hypersensitiveness by picking up the skin with force: the very moment you grab that area, if the appendix is in that triangle, the patient will not let you make any mistake and his hand will grab yours. If you find this area of acute hypersensitiveness of the skin, everything else pointing to a surgical condition, it is almost certain that you have acute appendicitis. Have found this sign in three cases since reading the articles of
and Livingstone.

I do not think it matters whether you cover the stump or not; I usually do not, but that is a personal preference. We can lay more stress on Murphy's old sequence of symptoms than perhaps any other picture we have; first, pain; second, nausea; third, temperature.

ANESTHESIA IN ACUTE APPENDICITIS

S. I. BLOOMHARDT, M. D., Phoenix, Ariz.

The choice of the anesthetic to be used in different surgical procedures is rather interesting and certainly one of very great importance. With all the complications, both medical and surgical, the personal equation of the patient, the nervous temperament and peculiar idiosyncrasy of every individual to be considered, the choice gives surgeon and anesthetist all the opportunity in the world to display their judgment—and what a valuable asset to one of our profession is judgment! How often we see the man with clever tongue or nimble fingers do his little immediate job so well! Then we get to thinking of his judgment in doing just what he did, and of his results, and we wonder.

I shall not go into detail about the advances made in anesthesia during the past ten or fifteen years, but I can say that we do not, as yet, have a perfect anesthetic and I am sorry that we do not, but hopeful that in the near future we may have.

Chloroform has been practically abandoned in this country, for its dangers are well known. Ether is no longer considered the ideal anesthetic for the general surgeon, because pulmonary and gastro-intestinal complications after operation are frequent and so often disastrous to convalescence or even recovery. Besides, its taste, smell and post-operative effects are obnoxious to the patient. Its administration to the badly injured or seriously ill patient requiring a major operation, may be the deciding factor of danger and its use in minor surgery is certainly no longer justifiable. In fact, it has gotten to the point where the public, in many localities, have been educated and will not consent to its use, and the patient comes to the surgeon questioning about the kind of anesthetic which is to be administered. It is a standing joke among the surgeons of New York whose patients are among the wealthiest, that the average patient will permit any one of a dozen surgeons to operate, but demands and will have none other than one anesthetist. Incidentally, his fee is $500 or nothing.

Nitrous oxide and oxygen anesthesia has supplanted ether in many clinics, for its advantages over ether are many. However it may be a very dangerous anesthetic in unskilled and untrained hands, for, unquestionably, there have been many deaths which may be attributed to its use. But, statistically, it has been shown that the high mortality has been due to its use by the untrained man. As a profession we are too prone to do this—just deciding that, since we can give ether, or perhaps chloroform, we also can give nitrous oxide and oxygen. The result is that this man or that tries, and he does not know his machine, his narrow margin between life and death.

And then we have ethylene, the last of our gases to be used generally, although it was first used, I believe, some forty years ago. Gwathmey has shown—and I believe

the other leading anesthetists agree—that the margin of safety afforded by ethylene is greater than that of nitrous oxide and oxygen, nitrous oxide and oxygen plus ether, or ether alone. Relaxation is rather difficult to obtain with nitrous oxide and oxygen, easier with ethylene. Iu 1923, Drs. Luckhardt and Carter, after much experimenting with ethylene gas, suggested its use as a surgical anesthetic. Dean Lewis and W. E. Brown, I think, were the first to employ it, believing it to be a safe and satisfactory anesthetic. And then, as with so many new things, we became over-enthusiastic and at last thought we had the perfect anesthetic. It is not that, but a very valuable addition to what we have had in the past. The gas was known to be very explosive, and this was emphasized by the early workers. There have been some explosions which were due to carelessness and some due to static spark, which was little understood at first, and no safeguards were employed in the machines to cope with this danger. The modern gas machines are made to carry off the static electricity and ground it, so that the danger of an explosion from the static spark is nil. It is unsafe to use a cautery or free flame of any kind in the operating room during the administration of ethylene. Another objection to its use is its disagreeable, sweetish, sorghum odor. We get around this by starting and finishing the anesthetic with nitrous oxide and oxygen. The operating team and nurse soon become accustomed to it. Different patients react differently to it as to post-operative effects, and it is an open debate as to the amount of post-operative toxicity in comparing nitrous oxide and oxygen, and ethylene. The personal equation of the administrator and the economic equation both have influence, because, by rebreathing a great deal, we can administer the gas more cheaply but with more toxicity. The advantages are many: the ease and quickness of induction, the prompt awakening after operation, relaxation which will permit major abdominal operations and the reduction of fractures of long bones and dislocation of larger joints. Sometimes ether must be added to get this to the full, but a cooperation between anesthetist and surgeon is most important to get this deep relaxation. Ethylene does not stimulate the respiratory center and, hence, the respirations are smooth and regular and resemble those of normal sleep. The skin is closed and dry and the patient's color remains good if the proper mixture is used. There is no extensive secretion of mucus during the period of anesthesia. The awakening from ethylene anesthesia is prompt and without too much excitement. As it has very little effect upon the blood pressure it is especially valuable as an anesthetic for the badly injured. I have elucidated a little fully on ethylene because it is the newest gas. Personally, I prefer that the surgeon make the choice of gas, and I frequently find, when one does not give the desired results in a patient that the other will.

As to local anesthesia, the idea of performing painless operations on a conscious patient is probably as old as any other modern development. The recent wide-spread use of local anesthesia is based on the discovery of the anesthetic properties of cocaine (well known to Indian healers), its isolation from cocoa leaves and its first use in eye surgery by Koller in 1884. This date marks the beginning of rapid development, mainly in two directions: The technique was evolved step by step; careful anatomical studies revealed exact landmarks for approaching nerve trunks; methods of infiltration, peripheral and central nerve blocks were described. The development of the pharmacological side of the question has not been so rapid and is by no means complete. Yet important steps may be noted here. Cocaine, except for surface anesthesia, has been replaced by the much less toxic novocain. The object of further development would be to find a still less toxic and more active anesthetic, that will alleviate post-operative pain for at least the first hours after operation. But in local anesthesia we should have the patient's age and psychic state, in addition to the absence of localized or generalized infection, to consider. Its advantages are: the diminution of post-operative pulmonary complications; the absence of heart, liver and kidney damage; the diminution of gastro-intestinal and bladder paralysis; and the possible co-operation of the patient. But will you not agree with me that the relationship between local and general anesthesia is not that of rivalry? We are so prone to become the slaves of a hobby or set idea; but, after all, the brand of a big man in contradistinction to the small man, is broad mindedness and common sense. Both local and general anesthesia have their advantages in certain types of patients and certain regions of the body, and, surely, they can be combined to a great advantage, especially in abdominal surgery.

Thus we have had a rather sketchy review of our more used anesthetics of the present period. Now, given an acute lower right-sided abdomen; the patient already

somewhat toxic; the muscles in their characteristic rigid spasm; the intestine, with its characteristic natural knowledge of protectiveness, quieter than usual (unless some over ambitious doctor has administered salts or oil, or both): what procedure should we follow in regard to choice of anesthetic? The patient has suffered great pain, is nervous and apprehensive, and may not be a good subject for purely local anesthesia. We know the dangers of chloroform. Ether is unpleasant, is very toxic, has a tendency to cause post-operative pulmonary and gastro-intestinal complications (particularly the latter because, to begin with, the gastro-intestinal tract has been grossly insulted). So will you not agree that the following procedures have proved to be the most successful? First, local anesthesia if the patient is mentally so constituted. Second, general anesthesia of either ethylene or nitrous oxide and oxygen-plus, possibly, a little ether if there are no pulmonary complications. Third, even better, the general can be combined with an abdominal infiltration of novocain or with an abdominal wall block, which, if well done, not only anesthetizes the incision but relaxes the rigid and spastic abdominal wall. The general anesthesia, therefore, need be really only an analgesia or exceedingly light anesthesia. About five-sixths to nine-tenths of the general anesthetic is saved in this way, you do not use sufficient local to increase the already existing toxicity of an acute abdomen, nor do you use enough general to endanger the patient's life (even though you may have a great fear of gas anesthesia) nor to increase the toxicity from the gas. And consider the shock, whether peripheral nerve or psychic, and sometimes I think the latter more damaging than the former, at least, it is more lasting. With a local infiltration or local block, you are cutting off the nerve impulses, which nothing but really a dangerously deep anesthesia can do, and hence there is a reduction of shock. With the light gas anesthesia you cut off the mental shock—the extreme fear, which any of you doing local work know, and which does not seem to harm the patient immediately but which lengthens convalescence. To be sure, if you have the phlegmatic-stoical type of patient, unexcitable and unemotional, use your local. Having been assistant attending surgeon to several of the large closed staff hospitals back East, I naturally got a great many of the acute lower abdomens to do, as the chiefs did the clinics and not many of the emergencies. Always having been interested in anesthesia along with the surgical side, the resident doctors and I would study and chart and try the different anesthetics and methods. Certainly the combined method, from the standpoint of ease of operating, from the standpoint of safety, from the standpoint of post-operative sequelae, and from the standpoint of convalescence, in our limited judgment seemed to prove the best.

In the series of acute appendix cases studied at this meeting, of the operative cases there were but two post-operative pneumonias, and both were following ether administration.

SUMMARY

1. Anesthesia has taken its place as an important and exacting part of surgery, and the choice of the anesthetic is a personal equation, requiring skill and judgment of surgeon and anesthetist.

2. Chloroform has been practically abandoned in this country on account of its dangers.

3. Ether is no longer considered the ideal anesthetic.

4. The gases have supplanted ether in our better clinics, but, at times, are most effective when a little ether vapor is combined with them. The choice of gas is often difficult, but certainly it is best to be equipped with both so that, if one is not efficacious, the other may be.

5. Local, in some cases, has its advantages; but the type of patient, the surgical condition, and the region of the body to be operated, should be considered. Also, the skill of the administration is an important element.

6. Of the pneumonia deaths following operations of acute appendicitis, in the series studied tonight, both followed ether administrations.

7. In acute appendicitis the anesthetic of choice is (1) local, either infiltration or block; (2) nitrous oxide and oxygen, or ethylene; (3) nitrous oxide and oxygen, or ethylene, plus ether; (4) possibly preferable, gas analgesia with novocain infiltration or novocain block.

GENERAL DISCUSSION

DR. G. M. BROCKWAY, speaking of the observations of the infrequency of appendicitis in a very young children, and of foreign bodies in the appendix, recalled a case where appendix was removed from a three year old child and there was a pin protruding from the end of the appendix. He also spoke of the possibility of confusing the symptoms of urinary calculus with those of appendicitis, reciting a case where the symptoms were more like those of calculus, with blood in the urine and absence of muscle rigidity and nausea. Operation showed a gangrenous appendix.

THREE CASES OF SCARLET FEVER OF UNUSUAL INTEREST OCCURRING IN THE SAME FAMILY.

J. A. RAWLINGS, M. D., El Paso, Texas

I wish to report three rather interesting and unusual cases of Scarlet Fever that have recently come under my observation:

The first case is one of anaphylaxis occurring in a boy of ten, one of a family of eight children, the eldest sixteen years and the youngest nine months. The family lived in a very small house, where it was impossible to segregate properly the sick from the well; besides, reduced finances forced the mother to have the care of both the sick and the well. The first case of scarlet fever occurred in the eldest child, a sixteen year old boy, on Dec. 7, 1926. The case was then in the hands of another physician who gave the boy a curative dose of scarlet fever antitoxin, and the other seven children were given prophylactic doses.

The boy who received the curative dose had a mild attack of the disease without complications. In about three weeks, three other children came down, but had mild attacks. On January 7th, a boy of ten was stricken and was seen by the attending physician the following day. He appeared much sicker than the others, with a temperature of 104, bad throat and pronounced rash, so the doctor thought best to give a curative dose. This was given at one o'clock. In less than half an hour the boy showed signs of anaphylactic shock, with great difficulty in breathing, rapid pulse, cyanosis and signs of pulmonary edema. The physician was called back and, at once, gave the boy a hypodermic of atropine, and adrenalin by mouth. The boy vomited and seemed relieved, so the doctor left but later had a phone message saying the boy was much worse. It was then that the doctor asked me to see the case. We hurried to the bedside but the boy passed away just as we reached the house. The length of time elapsing between the giving of the antitoxin and death was a little more than three hours. Later, by questioning the mother it was found that the boy had an attack of asthma when about four years of age.

The doctor did not see the family again for several days, so that, when another child was taken sick, he asked me to take charge, which I did.

This case proved to be a two and a half year old boy whom the family thought had escaped the disease. When seen, this child had a red throat, some cough and coryza and a temperature of 104, but no rash developed and on the second day the urine showed blood macroscopically and, upon microscopic examination, showed all kinds of casts, much blood, and two plus albumen. The boy was having an acute and severe hemorrhagic nephritis. My belief is that this boy had a mild case of scarlet fever and, in the absence of the doctor, had been overlooked by the family. In no other way can I explain the severe nephritis. Under appropriate treatment he is now making a good recovery.

A fourteen year old girl, who was one of the three who had the disease, as already mentioned, was also found to be suffering from a severe nephritis and was ordered to bed and placed on appropriate treatment. Because she did not appear very ill, the mother could not see the importance of keeping her in bed, so, one day when the girl complained of a severe headache, the mother allowed her to get up and dress. In fact, I found later that she was not following directions well at all. So, on this day, about eleven a. m., twenty-three days after her initial symptoms, she developed severe convulsions and became somewhat comatose. My associate, Dr. Harry Leigh, saw the patient and gave two doses, 2 cc. each, of fifty per cent magnesium sulfate intramuscularly, about one hour apart. This controlled the convulsions. The girl was taken to a hospital where the magnesium sulfate was continued at eight to twelve hour intervals for thirty-six hours, with hot packs and diuretics. No more convulsions occurred and the girl soon showed marked improvement. At this writing, three weeks later, the urine is nearly clear of casts and albumen. In the beginning, both this girl's urine and that of her younger brother, contained much blood and albumen with numerous casts, especially granular.

The lessons to be learned from these cases are several:

(1) The curative dose of antitoxin is unquestionably beneficial, cutting the disease down more than one-half in time and severity, and practically doing away with complications; but, like diphtheria antitoxin, it must be given in the first forty-eight hours of the disease to be most effective.

(2) Prophylactic doses protect for only a few days, perhaps a week or ten days at best, and their use is questionable.

(3) Both curative and prophylactic doses, in nearly all instances, cause severe horse serum reaction, starting in from four to seven days, sometimes later. This anaphylactic shock may be dangerous and

sometimes fatal. In nearly all cases there is a high temperature, marked general adenitis, arthritis, urticaria and intense discomfort and itching. Usually these symptoms are transitory and pass away within two to five days, without serious result. In a very recent article just published, A. R. Dochez recommends that, because of these untoward symptoms following the prophylactic dose, and because of its questionable value, it is best to wait until the child shows positive signs of the disease and then give a curative dose. But he also believes that even the mildest cases should have the curative dose in order to prevent complications.

(4) Mild cases of scarlet fever, so mild in fact that they are often overlooked, as in the case of the two year old boy, may be followed by a severe nephritis. These types, passing undiagnosed and undetected, are the dangerous types that spread the disease.

(5) Be guarded in giving any horse serum antitoxin to any individual who has had the serum before, or who has a history of hay fever, asthma or urticaria. Give a small amount into the skin and await results before giving the whole dose. My experience is that both tetanus and scarlet fever serums produce reactions more often and of a more serious character than does diphtheria antitoxin.

(6) I believe we have a most helpful agent in magnesium sulfate to be given either intravenously or intramuscularly in the treatment of uremic convulsions following nephritis either of the ordinary type or that due to pregnancy.

(7) In combatting the anaphylaxis due to these horse serums our best agents are adrenalin and atropine given in large doses and frequently repeated.

DISCUSSION

DR. E. W. RHEINHEIMER stated that he was a strong disciple of scarlet fever serum. He cited a case with marked symptoms,—with a temperature of 104.5 at 11 a. m., when curative dose was administered. At 8 p. m. temperature was 99, the next day normal, and remained so. In every case in which he has used it the temperature has been normal the following day. However, serum sickness developed in every case except one. He asked the question, "Should a child who has had serum sickness receive toxin-antitoxin for diphtheria?"

DR. R. A. WILSON stated that after the broad experience the City Health Department has had with scarlet fever serum, he regards it as absolutely specific and that they have had no untoward results from its use. He has abandoned the prophylactic dose. The City Health Department has administered 35,000 doses of toxin-antitoxin with no record of anaphylactic reaction.

DR. HARRY LEIGH brought out that the serum albumen in diphtheritic antitoxin is almost entirely eliminated and that nothing more than serum globulin remains. This is not true of the scarlet fever antitoxin, which accounts for the higher percentage of serum sickness when scarlet fever antitoxin is used.

DR. E. A. DUNCAN warned against using antitoxin in a sensitized patient.

In closing, DR. RAWLINGS stated that he believes toxin-antitoxin can be safely given to a child who has previously had serum sickness. He gave history of an epidemic of diphtheria in Kansas City in 1894. During the epidemic he used antitoxin for the first time; it was then made in powder form. Thirty years ago he administered a prophylactic dose of diphtheria antitoxin to his wife, and she is sensitive to serum at this time.

A CASE OF AMEBIC CYSTITIS

E. B. ROGERS, M. D.
El Paso, Texas.

During the month of June, 1925, a middle aged bachelor consulted me for an affection of the bladder. According to his history the trouble had begun some three or four months previously with frequent urination, accompanied by burning pain and tenesmus. These symptoms had gradually increased in severity until at times he would have to empty his bladder once every fifteen to thirty minutes, night and day, and the tenesmus and pain became almost intolerable. For about a month he had passed blood in varying amounts, at intervals large quantities with some small clots.

Physically, aside from the bladder condition, he presented nothing abnormal. The urine was alkaline, 1020, contained much albumin and blood and a smaller amount of pus. He had diagnosed his own case as gonorrhea and had been using drugstore remedies. A slide examination did not show gonococci. On the next day the urinalysis was unchanged except that there were plugs of mucus containing pus, blood and triple phosphate crystals. Prostatic massage gave pus, which did not show gonococci. Cultures from the prostatic pus and from the bladder urine failed to produce a growth on agar. Daily specimens showed a rapid decrease in the amount of blood, and at the same time the pus apparently increased so that on the third day the blood and pus were about equal in amount. On the fourth day the pus predominated. Since the only organisms ordinarily found in a non-fetid pyuria, that fail to grow on agar, are the gonococcus and the tubercle bacillus, a careful search was made for the latter, but without result. Two glass and three glass tests all gave about the same amount of pus.

Ten days after the first examination there was a sudden increase in the hematuria, which subsided like the one described. A cystoscopic examination showed only an acutely inflamed bladder, most marked in

the area of the trigone. Just within the vesical sphincter was a point of hemorrhage where arterial blood was spouting up like an artesian well. The field was so quickly obscured that the possibility of a small ulcer could not be determined. The ureteral catheterizations gave a trace of albumin on each side but no pus or blood.

The case was at first placed on medicinal treatment; diet, with increased fluids, hexamethylene with urinary acidifiers, and bladder sedatives. It might be noted that sodium benzoate failed and the urine could not be acidified except with unusually large quantities of acid sodium phosphate. After a few days local treatment was begun in the form of daily injections of one ounce of one per cent protargol into the bladder. After about a month a single clear specimen of urine was obtained but on the next day the blood and pus had returned. During the second month endoscopic treatments of ten per cent silver nitrate were applied once a week to the bleeding area at the vesical neck.

The subjective symptoms had been considerably relieved, but the intermittent attacks of hematuria persisted and the amount of pus varied greatly from time to time, being heaviest immediately following the hematuria. After the urine had stood in a glass for a time there would form in the bottom, a glairy, mucoid clot one-half to one inch in thickness which would be white if only pus was present or pink to red with varying amounts of blood. During a period of hemorrhage the centrifuged specimen would present an almost unbroken field of red cells. In three or four days the picture would change to pus with only a few red cells. Then the pus would diminish day by day until the next hemorrhage came on. In September, during a more protracted pyuria stage, there were noted numbers of cells that had all the appearance of amebic cysts. They were round, immotile, encapsulated by a heavy membrane, and contained two to four cell inclusions. The protoplasm was slightly granular and occasionally contained small vacuoles. Stained specimens gave the typical appearance of cysts and definitely excluded the round epithelial cell that often appears as a desquamation product in the later stages of cystitis. After studying specimens for a week in conjunction with my office colleagues we decided that the cells were amebae in the resting stage.

Treatment was now changed to a daily injection of emetine into the bladder. Beginning with one grain in one ounce of sterile water the dose was increased to two grains in one and one-half ounces of water and continued for two weeks. The symptoms were relieved, and the pus gradually diminished until it disappeared altogether. No amebae were found after this time and the urine culture was still negative. The urine remained clear and the patient seemed to be permanently relieved. Some months later he moved to California and has not since been heard from.

The diagnosis of amebic infection of the bladder has, in the past, invited much criticism. Dobell in his work on "Amebae Living in Man," cites twelve cases reported by various authors, and comments unpleasantly. One case he admits as "probably authentic," as he had known the worker personally. It seems as though he felt the condition to be impossible because he had not personally seen a case. Cases have now been reported by Kartulis, Posner, Walton, Macfie, Allen J. Smith, Petzetakis and others. Amebae have been found in almost every other part of the human body, why not in the bladder? No doubt cases are overlooked because of failure to follow up each case with the laboratory technic necessary to stamp the condition as a non-bacterial infection, which should lead one to search for protozoa.

Every case of amebic cystitis should have a demonstrable etiology, since this cystitis as well as other forms must be secondary to a focus of infection elsewhere in the body. The case here reported had no history of dysentery and no amebae were found in his stool on a single examination. However, since he belongs mentally with the "submerged one-tenth," the history may not be reliable. He lived with a brother-in-law who had had for years a chronic amebiasis.

The symptoms that might lead one to think of amebic cystitis are frequency, both day and night (pollakiuria), severity of the tenesmus and pain, and alternating hematuria and pyuria. Then when no organisms can be found in the stained specimen and cultures show no growth, search should be made for protozoa. The diagnosis of amebic cystitis is made in this case because of these symptoms and laboratory findings; because of the constant presence of cells that had both the histological and the staining characteristics of endameba histolytica; and because of the therapeutic test,—the organism and the symptoms disappearing and the patient recovering after treatment with emetine. Usually the treatment has been by hypodermic injections of emetine. I can find no record of any case having

been treated by injections of emetine into the bladder.

DISCUSSION

DR. G. WERLEY asked why the amebae were at the rest stage in this case. It was his opinion that the strongest point in favor of a diagnosis of amebic cystitis was due the fact that emetine produced the cure. The asbence of motile amebae and the fact that all observed were encysted, strongly suggested to him the possibility of these being blastomycetes.

DR. K. D. LYNCH would not believe that relief from emetine treatment proved the diagnosis. The sudden absence of pus and the clear urine for one day, to be followed by pus on subsequent days, suggested the possibility of a fistulous communication between the bladder and rectum.

MAJOR CRAFT believed that amebic infection of the bowel sufficient to produce erosion of the bladder would be accompanied by sufficiently marked symptoms as to be outstanding.

DR. F. D. GARRETT stated that the presence of amebae in the urine was not conclusive proof that they were the etiological factor in the cystitis, he believing that too much importance is attached to the mere finding of amebae, as often more detailed study will prove they are not pathogenic. In his work, unless there are gasto-intestinal symptoms he does not make a diagnosis of amebiasis on the mere presence of amebae in the stools.

DR. HUGH CROUSE has gotten good results from stovarsol and calcium carbonate intravenously, in amebic cystitis.

DR. JAMES VANCE thought there was an unusual amount of pus present for so small a lesion as Dr. Rogers described, and was rather inclined to feel as Dr. Lynch,—possibility of an existing fistula.

DR. E. B. ROGERS: I do not believe it follows, as mentioned by Dr. Werley, that if the condition in the bladder is an acute process there should be found moving, vegetative amebae in the pus. I have repeatedly found cases of chronic amebic colitis having acute exacerbations, with blood, pus and mucus but only amebic cysts in the stools. Half of the reported cases of amebic cystitis had only cysts, no moving forms having been found. Of course the amebae burrowing in the mucosa must be motile but on leaving the membrane they at once assume the precystic or the cystic stage. There is a possibility of mistaking the amebic cyst for blastomycetes if microscopic examination alone be depended upon. Even then some budding forms should be found and the organism should be grown on agar. The cystoscopic appearance of the bladder mucosa should differ from the congestion of a simple acute cystitis; there should be blastomycetic nodules and thickenings of the mucosa or ulcerations. Such a form of cystitis has not been described apart from generalized blastomycosis, and ninety per cent of these cases die.

A diagnosis of cystitis due to amebic cysts will always be open to doubt and criticism. I cannot explain the rapid changes in the amount of pus. Had it been due to a fistula, or the evacuation of an abscess or diverticulm it would seem that some organism, such as the colon bacillus should have been present. I am sorry that I have no permanent specimens. After beginning the emetine treatment the cysts disappeared before I realized that the opportunity of getting them had passed.

YAVAPAI COUNTY MEDICAL SOCIETY'S BI-MONTHLY POSTGRADUATE STUDY

At the regular bi-monthly joint meeting of the Yavapai County Medical Society (Arizona), and the Fort Whipple Hospital Staff, held at the Yavapai Club, Prescott, Ariz., February 1st, the three groups showed one hundred per cent attendance, as follows:—Group I, Drs. Brooks, Allee, McWhirt, DeWitt, Seibert; Group II, Drs. Looney, Yount, Loewy, Gatterdam, Bassett, McClarty, Matschke; Group III, Drs. Benedict, Southworth, Allen, Carhart, Jones, Devine, Flinn. Drs. Buck, Herrick and Rene were judges.

Group II had been assigned Cabot's Case No. 12493 (Boston M. & S. Jour., Dec. 9, 1926).

Group III had been assigned Cabot's Case No. 12411, (Boston M. & S. Jour., Oct. 14, 1926).

Mimeographed copies of the histories of these cases were distributed to each member of the society and staff, withholding the discussions and the necropsy findings.

On behalf of Group II, DR. R. N. LOONEY read the history of their case, as follows:

A German widow sixty years old entered August 10 complaining of jaundice. When she was nineteen she had an attack of jaundice with nausea and abdominal pain, not localized. She was in bed two weeks and out of work six weeks, with no severe pain or high temperature. When she was 42 she had attacks of sharp right upper quadrant pain of sudden onset lasting three hours, accompanied by nausea, relieved by "injections." The attacks occurred at any time of day. After them there was tenderness for a week or two. She had four of these attacks in the next ten years. When she was 52 her husband died suddenly. She was ill with another attack, with jaundice for the first time since the attack in her girlhod. After this she felt very well until ten weeks before admission. Then she lost appetite, especially for meat. When starting to eat breakfast she felt weak, with fullness in the epigastrium relieved by soda, milk and belching of gas. Six weeks before admission she noticed that her chest was yellow. The color had now spread to her face and arms and had grown more intense. She was in bed two weeks. For six weeks she had had difficult and painful micturition. For five weeks she had had edema of both legs. Four weeks before admission she began to vomit, at times coffee ground material but usually greenish, not bloody. The vomiting occurred especially on standing up. There was pain in the lower right quadrant, relieved by moving the bowels. Her appetite was poor in the morning, fair in the afternoon. The stools were loose, yellow and clay colored. The belching of gas and passage of gas had increased. Ten days before admission her hemorrhoids came outside and were replaced with great difficulty. She was more dyspneic than usual. Two days before admission she had a real chill. For two days her urine had been reddish brown. F. H. and P. H. of no importance.

Examination showed a well nourished woman with deeply jaundiced skin and sclerae. The teeth were all false. The heart and lungs were normal. There was tenderness in the left lower quadrant. Pelvic examination showed a lacerated perineum, cystocele and rectocele; no other abnormalities. Rectal examination showed the sphincter lax, several inflamed hemorrhoids. There was slight pitting edema of both ankles. The amount of urine is not recorded, specific gravity 1.010 to 1.020, red at one of four examinations, a very slight trace to the slightest pos-

sible trace of albumin at four of five, bile at all of five sediment examinations, occasional leucocytes at two, occasional red cells at one. The blood is not recorded, except leucocytes 9,000 to 10,000, hemoglobin 85 per cent, 3,200,000 reds. A Wassermann was negative. Icterus index 90. Bleeding time 5 minutes, clotting time 8½ minutes. Van den Bergh immediate reaction was positive. The stool showed bile. X-ray showed no evidence of organic disease of the stomach or duodenum. The stomach emptied very rapidly.

Dr. Chester M. Jones, in giving his diagnosis, said, "I am unable to explain the left lower quadrant pain and tenderness."

The patient was given 10 cubic centimeters of 6 per cent calcium chloride at 10 a. m. and 9 p. m., August 15.

August 16 operation was done. She made a good ether recovery and was comfortable two days later. The drainage was not excessive. The morning of August 19 the temperature began to rise. There was profuse drainage of bile stained serum from the wound. The patient vomited a great deal that day and in the evening looked very ill. She failed rapidly during the night. The morning of August 20 there were many coarse bubbling rales throughout both lungs.

The temperature, which was 101 deg. the evening before dropped to normal, but the pulse rose to 110. That noon she died.

DR. R. N. LOONEY: This woman was of the cancer age. Did she have cancer of the pancreas, bile tract, stomach or elsewhere? Or was the trouble due to gallstones with chronic pacreatitis and septicemia following operation? Our group has agreed on a diagnosis, which will be stated by our last speaker.

DR. E. A. GATTERDAM presented the first discussion, summary of which is as follows: The essential point in this case is that the patient is a woman of cancer age with pain in the upper right quadrant, requiring morphine for relief. There are a number of things to think about, such as appendix, kidney, gall bladder, stomach, duodenum, liver, and, in a woman, ovaries and tubes. The symptoms in this patient began in early life (age 19), with jaundice and pain, so naturally we think of the gallbladder and liver. After several attacks without jaundice, she has another one (age 52) with pain and jaundice, possibly another gallbladder attack. In person of this age, pain in that region with tenderness and jaundice probably means more than ordinary gallbladder disease or stones; it is the age when carcinoma is apt to occur. In recalling the anatomy of the gallbladder, we remember that it drains into the common duct which first joins the pancreatic duct and then empties into duodenum. This is a natural point for stone to lodge, and blocking the duct at this point backs infection into the pancreas, resulting in chronic pancreatitis.

I once heard Dr. Mayo say that, given a case of this age, with pain in the upper right quadrant, referred to the back, with jaundice or vomiting later followed by pain and jaundice, he would make a diagnosis of carcinoma of the gallbladder or duct over the telephone. In carcinoma of this region, the most common site is carcinoma of the stomach. Carcinoma of the duodenum is very rare as a primary lesion. Carcinoma of the gallbladder or common duct is quite common following repeated atacks of gallbladder disease or lithiasis. In this patient, we need to consider whether there is carcinoma of the stomach, gallbladder or pancreas. The only evidence of stomach cancer is the uncertain record of bloody vomiting. The x-ray examination was negative except for rapid emptying, which may occur from irritation about the duodenum, e. g., duodenal ulcer, gall-bladder disease with adhesions, enlargement of the head of the pancreas. The head of the pancreas, when enlarged, may encircle the duodenum or common duct. There are probably several factors in this case. It was apparently considered a surgical case and probably operated without positive diagnosis. The surgeon probably considered stones to be present, with possibly other complications. He thought that removal of stones would be indicated, and if chronic pancreatitis was present, this would be benefitted, and if cancer were found, operation would do some good.

In disease of the pancreas, we remember that any enlargement will cause obstruction of the duct, just as stone in the duct would do. In tumors of the pancreas, carcinoma is the first thought; in that condition, the onset is fairly rapid, with emaciation, loss of appetite, clay colored stools and general symptoms of carcinoma; this patient gives a long history which does not look like carcinoma of the head of the pancreas.

Acute pancreatitis is much more sudden than this patient's attacks, with shock, vomiting and usually death in a short time. With regard to chronic pancreatitis, we look for this to follow gallbladder disease, or blocking of the duct at the ampulla of Vater. In this case, with history of pain, relieved by injections, and jaundice, it would be consistent with infection of the gallbladder, with stasis, and then stone formation. Undoubtedly, at operation, they found stones in this gallbladder, with obstruction in the common duct.

Hydrops of the gallbladder occurs when stone blocks the common duct, and when followed by infection, we have empyema of the gallbladder. There is nothing to indicate that in this case, except the long history.

Have not said anything about the kidney, because there is nothing in the case pointing to kidney stone, appendicitis, ovarian or tubal disease.

The pain in the lower left quadrant brings into question the ovary, the sigmoid or descending colon. There is nothing to point to cancer of the colon. Diverticulitis is a condition which we hear little about; usually the x-ray examination reveals it. Do not consider the lower left quadrant symptoms significant.

Our diagnosis, to be given by the final speaker, rests on gallbladder infection, in connection with which we must remember gallstones and secondary pancreatitis. We must also remember that carcinoma may stimulate gallstones or that stones may lead to development of carcinoma. The blood findings, showing markedly diminished red cells and practically normal hemoglobin occurs in carcinoma as well as perricious anemia.

DR BASSETT continued the discussion; his remarks, summarized, were:. Given a young woman with gall stone colic and repeated attacks during the following years, we must expect a certain amount of pathology in the bile tract. We have such a case; the history tells us that much and whether or not the immediate condition is an afermath of the previous attacks is to be determined.

Infectious jaundice is not a grave disease; it is a mild condition responding readily to treatment, but it leaves behind a fertile field for further trouble. Presuming this woman to have had an infectious jaundice and subsequent attacks of biliary colic, we assume that she has an infected gallbladder with stones, cholangitis and possibly chronic pancreatitis. It would not be unreasonable to assume some cirrhosis of the liver as a result.

The woman comes in with history of having been previously sick for several weeks; she was deeply

jaundiced with icteric index of 90, completely saturated with bile. A condition like that cannot exist very long without anemia which she had to a marked degree; bile acids in the circulation result in a grave anemia frequently fatal; they have hemolytic action, and not infrequently produce a picture like pernicious anemia. As a further result of biliary acids in the blood we have also explanation for the edema of the extremities and the painful micturition, this latter condition resulting from the irritant effect upon the bladder of the bile acids excreted in the urine. Icterus may or may not cause itching; this patient had no itching. They had to choose between hemolytic or obstructive jaundice; they performed Van den Bergh's reaction which indicated obstructive jaundice and they proceeded to operate. Whether the obstructive jaundice is due to gallstones or not, we cannot say. It may result from repeated attacks of cholecystitits, which this woman had. We cannot exclude carcinoma.

DR. LOEWY presented final conclusions: It has been truly said that persons seldom die from what ails them. We consider that this woman has had gallbladder disease for 18 years. At the age of 42, she suffered attacks without jaundice; gall stone blocks the passage and she comes in at 60 with obstruction; whether it is stone or tumor cannot be definitely differentiated. You may have carcinoma a long time and have it light up following infection. We can rule out stomach and duodenal lesions. Lesion of the head of the pancreas should be palpable.

Our diagnosis is obstruction due to cholelithiasis, with cholangitis and pancreatitis, but carcinoma cannot be definitely ruled out.

DR. W. E. McWHIRT then read the following discussion and autopsy report, from the Boston Med. & Surg. Jour. of Dec. 9, 1926:

DISCUSSION

BY ERNEST M. DALAND, M. D.

We notice that this woman complained of jaundice; she did not complain of pain.

That attack at nineteen was apparently an infectious jaundice, which probably had nothing to do with the present condition.

Apparently these attacks of pain were due to gallstones.

I do not know that "especially for meat" means anything. It does not to me.

This is a rather peculiar story, that she had this epigastric distress at the time of eating, but it was relieved by milk.

From her history we have a woman who apparently had an infectious jaundice as a girl, who had had numerous attacks of right upper quadrant pain sufficient to require injections, probably morphia. She had had jaundice once before during one of the attacks. The present attack had been on for six weeks, beginning with jaundice. There is not much said about pain along with the jaundice. She had some gastric upset, a certain amount of epigastric distress.

I cannot connect up the urinary symptoms with the rest of the story from anything I have heard so far.

Apparently these hemorrhoids were not due to anything higher up, because rectal examination was negative. The fact that she had had these hemorrhoids come out and that they had been replaced with difficulty, the fact that she had edema of the ankles, would point to some backing up of the circulation in the lower part of the abdomen.

She has a slight secondary anemia.

The icterus index is very high. This examination all points towards an obstructive jaundice rather than a hemolytic jaundice.

DR. JOHN D. CAMP: These films show no definite change that is significant. There is a deformity in the first portion of the duodenum, which if we were to rely altogether on the films I think should say was an ulcer. This was supplemented by fluoroscopic examination; and the deformity present on the films was probably not constant, as we have this negative report. That illustrates why we should not rely on x-ray films alone.

DR. DALAND: This woman had obviously had gallbladder disease in previous years, and I think that the most likely diagnosis in view of that is that she had a stone in the common duct. She did not complain of much pain during the six weeks that she had this jaundice. I think patients are pretty apt to have pain at the time the stone goes down into the common duct.

The other possibilities are a carcinoma of the gallbladder or a malignancy of the head of the pancreas.

The patient had a chill two days before she came. That might indicate some reaction in the liver such as would be given with a cholangitis. She had a normal white count, so that it is not apaprent that she had any severe gall bladder infection.

I think probably she had stone in the common duct. I doubt very much that she would have been explored so quickly if they had thought they were going to find malignancy.

DR. DALAND'S PRE-OPERATIVE DIAGNOSIS

Stone in the common duct.

PRE-OPERATIVE DIAGNOSIS

Stone in common duct.

OPERATION

Gas-ether. A seven-inch incision over the gallbladder. The gallbladder was distended and slightly thickened, but contained no stones. The stomach was normal. The kidneys and spleen felt normal. There was a definite hard swelling of the head of the pancreas, and the body and tail of the pancreas were slightly swollen. No stones were felt in the common duct. "The diagnosis is obviously carcinoma of the head of the pancreas." A cholecystogastrostomy was done in the same method as a gastroenterostomy, using two posterior and three anterior layers of number 0 continuous catgut. One cigarette wick.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Carcinoma of the pancreas.
Peritonitis?
Cholecystgastrostomy.

DR. ERNEST M. DALAND'S DIAGNOSIS

(1) Stone in the common duct.
(2) Carcinoma of the pnacreas?

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion.*
   Red atrophy of the liver.
2. *Secondary or terminal lesion*
   Icterus.
   Hemorrhage into lung tissue involving a large portion of each lung.
   Hemorrhage edema of the lungs.
   Slight arteriosclerosis.
3. *Historical landmarks*
   Slight chronic pleuritis, right.
   Obsolete tuberculosis of a bronchial gland.
   Operation wound.

DR. RICHARDSON: There was a well-marked general icterus. In the peritoneal cavity there was a small amount of thin bloody fluid but no peritonitis. The appendix was negative. The esophagus was negative.

The gallbladder was securely united with the stomach at a point about four centimeters above the pylorus. The part was in good condition, and the opening between the gallbladder and the stomach was patent. The gallbladder itself contained but little bile. The mucosa was smooth and the bileducts were free and negative. The intestines showed moderate congestion.

The pancreas macroscopically showed a definite lesions. There was no deformity and grossly there was no evidence of new growth. Microscopically the organ was negative.

The spleen weighed 170 grams; the tissue was dark brown red, elastic, congested. The adrenals, kidneys, uterus and adnexa were negative.

Head. The meninges and vessels of Willis, the middle ears, pineal and pituitary glands were negative, and the brain, which weighed 1434 grams, showed no lesions.

The trachea and bronchi cointained much bloody, frothy fluid. The apices of the lungs were negative. There were no areas of consolidation, but the tissues of each lung over large areas was dark to blackish red and markedly infiltrated with blood. Large areas then of the lung tissue showed hemorrhage. The remaining portion of the lung tissue showed hemorrhagic edema. Hemorrhage of the lung, presumably associated with the icterus.

The heart was negative. The aorta and great branches showed a very moderate amount of fibrous sclerosis.

The liver weighed only 760 grams. That is pretty small for a liver—about half size. It was fairly symmetrical. There were a few adhesions between the upper part of the right lobe and the diaphragm, these somewhat vascular. The surface generally finely to coarsely granular with areas where it was fairly smooth. The tissue generally was leathery and was bile stained. The section surfaces were generally granular, with areas where they were dark reddish, fairly smooth and rather elastic. The anterior margin of the liver was rounded and finely to coarsely granular. And there we have a mixed picture, in some places resembling cirrhosis and in others a late stage of a toxic or infectious hepatitis. Macroscopically it certainly suggested that it might be a late stage of toxic or infectious hepatitis and microscopically it was considered to belong to the so-called yellow atrophy group, late stage.

DR. CABOT: There is no organ of the body in which I have seen such frequent disagreements between surgeons and pathologists as the pancreas. We are always hearing the surgeon found this or that in the pancreas, and when we come to necropsy Dr. Richardson refuses to find it.

Group III discussed the following case record, taken from the Boston Med. & Surg. Jour. of Oct. 14, 1926:

A widowed American laundress 61 years old entered August 18 for the first time complaining of hematemesis.

Seven months before admission she suddenly felt nauseated one evening and then brought up a fair amount of bright red blood. By the advice of her physicion she rested a week. Then she went back to work. Two months before admission she had a second similar attack with less blood, only half a pint and no nausea or feeling of weakness. She went back to work after two days. Eighteen days before admission, soon after being awakened by a fire alarm in the night, she brought up two quarts of bright red blood. She felt weak for a few days. Since that time she had been resting, but had no other symptoms. With one of the hemorrhages she had a "light" feeling in her head. She had tarry stools for two or three days after all the hemorrhages, but at no other time.

Her mother died of heart trouble in old age. Her first husband died of consumption after four years illness. The patient had never had children. All her life she had been quite healthy. She passed the menopause with no trouble at the age of 46. Her weight had been 205 pounds for the last ten years and was 203 eighteen days before admission.

Examination showed a very obese woman with clear pale skin and marked pallor of the mucous membranes especially the mouth. At the left apex posteriorly there was distant bronchial breathing. Voice sound, no resonance, not increased. Tactile fremitus diminished. The apex impulse of the heart was not found. The heart was enlarged at the arch and in all directions except downward. Percussion accuracy was impossible. The aortic second sound was accentuated and snapping. There was a systolic murmur over the left sternum, loudest at the aortic area. The heart sounds were distant. The blood pressure was 190/100-165/90. The liver edge was felt two centimeters below the costal margin. There was a mass (spleen?) descending with inspiration in the left upper quadrant 4 centimeters below the costal margin. A rounded pole like the spleen or kidney presented. At pelvic examination on the table the uterus filled the left fossa. It was not tender, but seemed larger than normal. (Fibroid)? Inspection showed a normal os. The fingers showed Heberden's nodes. The pupils and reflexes were normal. Fundus examination showed normal discs, sclerosis of the vessels, a small area of hemorrhage and exudate in the nasal side of the right disc.

The amount of urine is not recorded. The specific gravity was 1.021 to 1.020. The sediment showed many leucocytes at all of these examinations, rare red cells at one. Renal function 30 per cent, polynuclears 70 to 49 per cent, reds 2,800,000 to 4,100,000, marked achromia, some anisocytosis and poikilocytosis, no stippling or polychromatophilia; platelets seemed decreased in one of four smears, slightly increased in two later stained with cresyl blue. Reticulated cells .5 to 1 per cent. Clotting time 6-9 minutes. Clot retraction poor in three tubes, good in one. Non-protein nitrogen 30 mgn. Wassermann negative. Icterus index 2. Stools negative to guaiac at five examinations.

X-ray showed slight increase in the transverse diameter of the heart on the left and a slightly tortuous aorta. A barium meal and enema showed no definite evidence of organic diseases of the gastrointestinal tract. There was slight spasm in the signoid, but the various portions did not appear abnormal in contour.

The patient's color improved markedly. The increase in blood platelets over normal was notable. September 2 she was discharged.

After leaving the hospital she was followed in the out-patient department by the senior house officer. Her spleen and liver remained at least the same size as at discharge. At one observation a month after she left the hospital they were slightly larger. Two months after her discharge her weight had been reduced by twenty pounds. Her hemoglobin was 60 per cent and the red count 3, 720,000. The blood pressure was 200/90. In November she complained of slight dyspnea. She felt better if she took salts three times a week. At that time she felt well enough to begin working.

One morning in the middle of March, six months after her discharge, she was awakened in the night by a thunderstorm. She was nauseated, and after coughing a little brought up a big mouthful of bright red blood. The next day she had a tarry movement and felt weak. April 26 after breakfast she felt nauseated and brougrt up her breakfast mixed with what she thought was a mouthful of bright red blood. She had no bowel movement from time of her last hemorrhage until her admission to the hospital April 27. She felt weak and thirsty but did not remember fainting. Her memory was now very poor.

On examination she was well nourished, extremely pale, with a slight subicteric tint. The mucous membranes were markedly pale. The lungs were clear. The heart showed no enlargement to percussion. There was a soft apical systolic murmur. The abdomen was moderately obese, with questionable free fluid.

The liver edge was just felt. The spleen was questionable. Rectal examination was negative. The urine was normal in amount, specific gravity 1.018 to 1.020, the slightest possible trace of albumin at one of five examinations, rare to numerous leucocytes at all but one of five sediment examinations, rare red cells at one. Blood examination showed 6,800 to 4,600 leucocytes, 70 per cent polynuclears, hemoglobin 30 to 50 per cent, reds, 1,560,000 to 3,800,000, moderate achromia, poikilocytosis, anisocytosis, and occasional microcytes. The cells appeared smaller than normal, about one stippled cell per high power field, no nucleated red cells seen. Platelets apperaed normal, not increased. Reticulated cells 5 per cent to less than 0.1 per cent. Icterus index 3-4. Clotting time 9 minutes. Bleeding time 4 minutes. Wassermann negative. Stools: guaiac positive or very strongly positive at all of five tests to May 6, negative May 7, very strongly positive May 9, negative at three later tests. Rosenthal bromsulphalein test normal.

X-ray examination with a barium meal was made in the recumbent position only. There was slight flattening of the first portion of the duodenum. There were no filling defects to suggest organic disease of the stomach or duodenum.

Consultants: Internist. "I can see no indication for operation. Do not believe she has any type of primary blood condition. Spleen just palpable at costal margin. Liver down two fingers. Both liver and spleen smaller than in September. The most probable diagnosis seems to me to be malignancy of the stomach, in spite of negative x-rays and negative gastric history. Transfuse now and re-x-ray soon."

Surgeon: "I cannot make a diagnosis in this case. Malignancy does not seem probable. I cannot feel the spleen or liver. . . It would seem hardly justifiable to do a splenectomy on the evidence we have at present."

The temperature was 97.9 deg. to 100.4 deg., the pulse 70 to 111, the respiration not remarkable.

The patient had no more bleeding. May 5 surgical transfusion of 600 cubic centimeters of whole blood was done. A laryngologist advised against esophagoscopy.

May 20 the patient ate her supper as usual, and at the rounds at 6:45 was feeling very well. At 7:45 she suddenly brought up 450 cubic centimeters of fresh blood mixed with her supper. Ten minutes later the pulse was 96, the blood pressure 150/65. There was no evidence of shock. She was given a quarter grain of morphia. At 8:45 she vomited 400 cubic centimeters more of blood. She looked washed out, but was not in shock. May 21 she bled 500 cubic centimeters more. 600 cubic centimeters of blood was transfused. May 22 she vomited about 1000 cubic centimeters of bright red blood and two hours later 800 cubic centimeters more. After having the situation fully explained to them patient's relatives decided to have no more transfusions done. May 23 the patient again vomited six ounces of blood. May 24 she died.

DR. BENEDICT, captain of Group III, after summarizing the above record said that hematemesis is due to several causes; (1) swallowed blood which seems to be ruled out here; (2) disease of the esophagus which is considered to be the cause here; disease of the stomach, usually ulcer, of which there is little evidence in this case; disease of the duodenum, also usually accompanying symptoms, which are absent here; portal obstruction sometimes gives a profuse bleeding of this character, and we think that therein lies at least a part of this woman's trouble; acute febrile diseases somtimes gives profuse bleeding, which we think can be ruled out here; diseases of the blood may cause such bleeding and that phase will be discussed.

Profuse stomach hemorrhage will occur in one of three conditions; (1) gastrostaxis; (2) gastric ulcer; (3) cirrhosis of the liver, in which there is strong probability of producing the sort of hemorrhage we have. This woman had a high blood pressure, probably due to arteriosclerosis. She had a systolic murmur which may be due to heart lesion or the blood condition following the hemorrhages.

Group III will attempt to show that the evidence points to trouble in the liver, and that blood disease or disease of the spleen, while either may exist, is not of primary importance. The principal diagnosis will be cirrhosis of the liver with varix in the lower esophagus or upper end of stomach.

DR. ALLEN'S discussion, summarized, is as follows: The principal symptom is many and repeated hemorrhages. Outside of a little enlargement of the liver and spleen, that is the only diagnostic point. The blood conditions causing hemorrhage are better understood by reviewing certain points about the blood. The granular leucocytes and red cells are formed in the bone marrow; the lymphocytes come from the lymph glands; during the evolution of these cells, we have as antecedents of the red cells the erythroblasts, which are large nucleated forms, probably two or three times the size of the red cells; as evolution proceeds, this nucleus becomes shrunken and paler and the cell smaller, being called normoblasts at this stage. Later on the nucleus is extruded from the cell and we have the mature red cell. In the evolution of the granular leucocyte, we have the same process; originating from the bone marrow cells, we have, first, the myeloblasts, then the myelocytes, then the mature cells, eosinophiles, basophiles, or neutrophiles.

In blood diseases, owing to the stimulation of the marrow, and the migration of these cells into the blood stream, abnormalities in form, shape, staining, etc., occur. Anemia due to blood destruction is followed by marrow stimulation as the essential feature, and this occurs in the majority of blood diseases; exceptions are chlorosis and myelopathic anemia, or growths in the bone marrow substance which limit cell production. Another type of anemia which is essentially one of depressed blood formation, is the so-called aplastic anemia. In none of these three do you get marked bleeding.

We may divide the blood diseases into (1) those due to diminshed blood production; (2) blood destruction and (3) acute and chronic blood losses. Frequently, in diseases like perincious anemia, there is a stimulation of the blood producing cells and then the prognosis, or progress of the case depends on the balance between blood production and blood destruction. The most important of the anemias characterized by blood destruction is pernicious anemia, in which you have a variety of pictures, but there are one or two characteristics; you always have a plus color index, and you have many immature forms, characterized by changes in size, shape, etc.; wo do not have such a picture in this case.

In acute blood loss, after the first few hours, you have a marked leukocytsis which may run from 20,000 to 40,000, with a minus color index, which we have in this case; however, the leukopenia in this case would rule out anemia due to acute blood loss. In chronic blood loss, when the bleeding is continued over a long period, we have the picture presented by this patient. In such long continued loss, as we have here, there is a depressed function of the bone marrow; with this depressed function, changes in the blood picture different from acute blood loss occur; there is a leukopenia instead of leukocytosis and the blood pplatelets are diminshed. In reviewing the findings, there is nothing that

would not conform to chronic blood loss, and we believe the blood picture is due to that.

There is one other condition which we have not discussed, and that is Banti's disease or splenic anemia. The characteristics of this are marked increase in size of the spleen, a chlocrotic type of blood, with a minus index, and leukopenia. Therefore, we cannot rule out Banti's disease on the blood picture. This condition occurs in early adult life and the splenic enlargement is always noticeable. In this case the spleen, after being enlarged, has retrogressed in size and at the last examination there was no enlargement. So, Banti's disease is questionable, although frequently in that disease there is cirrhosis of the liver, and it may be that this is Banti's disease which has progressed to the stage of liver and splenic cirrhosis.

DR. FLINN'S discussion may be summarized as follows: This case is unique in one respect, as compared with any other case of our series for several years; the visiting and consulting staffs of the Massachusetts General Hospital, after studying it over for nine months, were apparently not able to make a definite diagnosis. In the light of this fact, Group III considers that it has considerable termerity in even suggesting a probable diagnosis. Another marked feature is that up until the time of the first hemorrhage, there was practically not a single symptom, and even after profuse bleeding for nine months, there are still very few symptoms.

In a general way, the case is remarkable in being practically symptomless; there are a few definite signs but no symptoms. It seems that the chief question becomes, "What disease or combination of diseases will produce these signs without symptoms whatever."

As pointed out, the chief presenting sign is profuse bleeding which we believe to be hematemesis, either from the lower end of the esophagus or the cardiac end of the stomach. In a hemorrhage of this size, quite a large vessel must have broken either an artery or vein. If this were an artery, the breaking of the vessel would probably be due to ulceration; if a vein, it is probably a varicosity. The chief cause of ulceration in this region would be gastric ulcer or malignancy, both of which we believe are ruled out by the absence of symptoms. The two chief causes of varicosity are obstruction in the portal circulation and some form of anemia. The last speaker went fully into the question of anemia and gave the conclusions of Group III.

We believe the question narrows down to, "What disease or group of diseases will cause portal obstruction and give practically no symptoms?" To understand the possible pathology, we must review the anatomy of the portal system. The portal vein carries the blood of the digestive system to the liver, and is formed by the union of four large veins; (1) gastric from the stomach; (2) spleenic from the spleen; (3) superior mesenteric, carrying blood from the small intestines, cecum, ascending and transverse portions of colon; (4) inferior mesenteric, from the descending colon, sigmoid and rectum. If we have obstruction in the portal system, we have a passive congestion all along the gastro-intestinal tract. There are two principal causes of portal congestion; (1) cirrhosis of the liver, and (2) thrombosis of the portal or splenic veins. In addition to the portal system, we also have a system of venous anastomosis, a compensatory circulation which is composed of very small veins but which, under portal back pressure, may become dilated and carry off the blood from the portal circulation. This compensatory system consists of anastomoses from the portal system through the left coronary vein of the stomach to the esophageal vein and from the esophageal vein to the azygos, intercostals and vena cava. When blood is forced through these anastomotic veins, what will happen? An intense varicosity will result at the lower end of the esophagus or upper end of the stomach.

The conception of Group III of this case is this: The patient died from hemorrhage from the upper end of stomach or the lower end of the esophagus. This bleeding was due to varicosities of the esphageal vein, caused by back pressure from the portal system through this anastomotic system. Our diagnosis is portal obstruction, due either to cirrhosis of the liver, with or without accompanying splenic anemia, or to thrombosis in the portal system.

DR. JONES read the discussion of Dr. Cabot and the nocropsy findings, as follows:

DR. CABOT: When hematemesis comes with no previous symptoms, as we say "out of a clear sky," cirrhosis of the liver is the commonest cause. Probably this will not be cirrhosis of the liver, but that is the right thing to think of as the commonest cause.

Two quarts of blood is a great deal, if it is so. It is very easy to get mixed up with stomach contents, etc.

We know from the war that people have lived after bringing up two quarts of blood. It is really striking how little these hemorrhages pulled her down. We could not have a case more illustrative of whatever turns out to be the cause of hematemesis in a person of perfectly good health, without any previous symptoms. Suppose one came to the bedside of a person who had just vomited two quarts of blood. Suppose one took the hemoglobin and found it normal. How soon should he expect it so go down? It takes forty-eight hours to get the blood diluted with the tissue fluids so that the patient is anemic. We saw that very often in the war. People lost a lot of blood, and yet the hemoglobin was perfectly normal.

Distant bronchial breathing on the left is much more significant that if it was on the right. Accuracy of thoracic percussion is always impossible, perhaps a little more here than usual.

Of course, if we are thinking of cirrhosis, as we are, and if this abdominal mass is spleen, that supports the diagnosis, because the spleen is usually enlarged in cirrhosis of the liver.

The fundus of the eye shows what we should expect with high tension at her age.

This is one of the cases of apparently fixed gravity of the urine, at a high point, which has no significance in my opinion. We are not told whether these were catheter specimens, so we shall disregard these statements as to the sediments. There was no albumin and no sugar, and I suppose nothing important in the sediment.

The leukocytes count is below normal, and that is characteristic of one disease which shows an enlarged spleen and sometimes other symptoms like these. Splenic anemia is what I had in mind,—or Banti's disease, which is indistinguishable from cirrhosis in many cases. There is no evidence of active regeneration on the part of the marrow, which is what strippling and polychromatophilia show. Cresyl blue is the stain we use for reticulated cells. That is normal here.

Let us sum up what we have so far. We have a woman who comes in for vomiting blood. We find a big liver and a big spleen, and I presume no ascites demonstrable. We have a high blood pressure, we have a big heart, we have a normal urine, we have an apparently secondary anemia of considerable severity. We have no evidence in the blood of any attempt at regeneration, I do not know why not. X-ray shows nothing of importance. We have ruled out so far as we can any organic disease like cancer or ulcer of the stomach.

What diagnosis did they make at the time of her stay in the hospital? I think they made a diagnosis

of Banti's disease or splenic anemia, because of the condition in the blood and the way the disease started. It is never a very satisfactory diagnosis. We are never sure that it is not cirrhosis of the liver.

The word "hypertrophic" is not one that is used now in speaking of cirrhosis. Alcoholic cirrhosis of the liver may give a big liver, a normal sized liver, or a small one. Biliary cirrrhosis, the so-called Hanot's disease, may give a large liver or one of normal size. So that the term "hypertrophic" has no significance. In most cases of cirrhosis that I have seen the liver has been big. There is overgrowth and fat and regenerative tissue, so that the whole organ is big.

The best diagnosis I can make is cirrhosis of the liver or Banti's disease, leaning toward the latter. I do not make any distinction between Banti's disease, and splenic anemia.

Blood pressure measurements in the out-patient department showed quite a big pulse pressure.

At the time of the thunderstorm probably most of the blood went down instead of coming up.

She came back to the hospital after her third hemorrhage.

NOTES ON THE SECOND PHYSICAL EXAMINATION

Nothing more is said about the left apex, which was queer before. It just blew away I suppose.

It is not likely that her heart got smaller. Hearts do not do that. Either the first percussion was wrong or the second was wrong or they were both wrong, as most percussion is.

It is pretty hard to tell about fluid sometimes in a thick-walled abdomen.

The anemia is a good deal worse than before, even though she brought up only a little blood. A lot must have gone the other way. One stippled cell per high power field is a good many. Five per cent reticulated cells is great increase over the normal.

The Rosenthal test is for liver function.

We have a bad slang phrase here, "blood condition." It means "blood disease," and we ought to say so.

The internist had good nerve to assert that cancer of the stomach is present when the x-ray is negative, but that has happened. Most malignancy which x-ray misses is on the posterior wall.

They wanted to see if they could see any varicose veins at the root of the esophagus. But the wise laryngologist knew the esophagoscopy has itself some risks. So the satisfaction of intellectual curiosity which we sometimes push too far was foiled here, I am glad to say.

I do not think it is gastric disease, either ulcer or cancer. I am still in doubt between the two I spoke of in the beginning. As she is a woman and probably did not have an alcoholic history I would rather assume Banti's disease than anything else. But I think the chances are it is something none of us has thought of at all.

This is a seven-foot x-ray plate, taken to show the actual size of the heart. The tortuous aorta means nothing. It used to be mistaken for aneurysm of the aorta in the early days of x-ray work. I should think the transverse diameter of the heart was big. As we have a high blood pressure I think there will be hypertension, having so far as I know no particular relation to the patient's death.

Miss Painter: The chest was twenty-seven and the total diamter of the heart was 14.2.

Dr. Cabot: 14.2 is more than half of 27. Therefore the heart is big. That is the rule here.

I do not see anything wrong with this x-ray of the lungs. The diaphragm and the bubble underneath are normal.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Cirrhosis of the liver.
Esophageal varices.
Hypertension.
Transfusions.

DR. RICHARD C. CABOT'S DIAGNOSIS

Banti's disease, or
Cirrhosis of the liver.
Esophageal or gastric varices.
Hypertension.
Hpertrophy and dilation of the heart.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesions*
   Cirrhosis of the liver.
2. *Secondary or terminal lesions.*
   Varices of esophagus and stomach.
   Anemia.
   Hemorrhage into gastro-intestinal tract.
   Edema of the lungs.
3. *Historical landmarks*
   Arteriosclerosis.
   Hypertrophy and dilatation of the heart.
   Slight chronic pleuritis, right.
   Chronic salpingitis.

DR. RICHARDSON: The skin and mucous membranes were very pale. There was no evidence of icterus. In the right cubital space was a short sutured wound. There was a large amount of subcutaneous fat. The peritoneal cavity was negative; no ascites. The appendix was negative except that the tip was bound down by old adhesions on the right side of the peritoneal cavity in the region of the tube and ovary; that is, there was more chronic salpingitis with the appendix tip bound down by the adhesions.

Along the lower two-thirds of the esophagus there were irregular rows of varices, and the tube contained much thin pale blood. The stomach also contained much thin blood and old blood-clot-like material, and there were some varices in the stomach, but not so marked as in the esophagus. The intestines showed rather thick, somewhat elastic walls, the vessels injected, but were otherwise negative. In the large intestine the fecal material was blackish red, apparently mixed with blood.

The liver was two centimeters above the costal border. The diaphragm on the right was at fourth rib, on the left at the fifth rib. That locates the liver, which weighed 1459 grams, if anything rather small for her.

There were a few plueral adhesions on the right apex. The lungs were pale, spongy, and showed much edema.

The heart weight 490 grams. That is considerably enlarged for her. The valves and the myocardium were negative. The coronaries showed a moderate amount of sclerosis, but were free and fairly capacious. The ascending thoracic aorta showed a very slight amount of fibrous sclerosis, but the rest of the aorta showed much fibrous and fibrocalcarous sclerosis, and in places areas of atheroma. The great branches of the aorta showed a slight amount of fibrous sclerosis. So that we have a hypertrophied and dilated heart and the amount of arteriosclerosis mentioned. There was but little blood in the heart cavities.

The liver weighed 1459 grams and showed frank cirrhosis. The peritoneum in the region of the liver showed a vascular meshwork.

The spleen weighed 600 grams, considerably enlarged, with smooth surface and elastic brown-red tissue.

The kidneys weighed 270 grams, the markings and tissue generally negative, except that they were very pale.

The uterus and adnexa were negative except for the chronic salpingitis mentioned.

Dr. Cabot: Suppose a cantankerous person wanted to maintain against you that that was Banti's disease, how would you differentiate it?

Dr. Rirchardson: The amount of cirrhosis with so-called Banti's disease, as far as we have seen it

here, is comparatively small in extent, with no such accompanying picture of cirrhosis, bleeding and varices.

Dr. Cabot: Many of the descriptions of Banti's disease speak of varices, the hemorrhages and the whole picture that we see here. I do not see how the distinction can be made between cirrhosis of the liver and Banti's disease.

Dr. Richardson: In the cases called Banti's disease here the outstanding things have been the enlarged spleen and anemia. The spleen in this case weighed 600 grams, and there was some increase in interstitial tissue. There was marked cirrhosis of the liver, and a spleen of that nature might go with it.

Dr. Cabot: The ordinary conception of Banti's disease is a trouble which starts with enlargement of the spleen. If that is removed it can be cured. If it is not removed there is progressive cirrhosis of the liver. It is a question how long the patient can live without hemorrhage cutting short life. But cirrhosis of the liver always has a big spleen too, and how big it shall be with straight cirrhosis and how small with Banti's disease there is no way of saying. We never shall get anywhere in this point of differential diagnosis on the basis of big spleen and ascites, because we have no conception of the cause of either disease. except I believe that alcohol has a good deal to with cirrhosis.

A Physician: In cases of cirrhosis are hemorrhages not so liable to occur in the hypertrohpic stage as in the atrophie stage?

Dr. Cabot: That is not my impression. It may be true. Your question implies the familiar idea that the cirrhotic liver starts big and gradually gets smaller, that the early stage is an enlarged liver and the later stage a small one. I do not believe there is any good evidence for that. Nobody has ever watched the liver get smaller. We find big livers at the end; we find small livers at the end; and I do not know that the liver goes through the stages of big, normal, small. Neither do I know of any particular relation of hemorrhage to the big livers and small livers.

Physician: Anatomically how do you differetiate Banti's disease from Gaucher's disease?

Dr. Richardson: They probably belong to the same group. In Gaucher's cases there is a very large spleen and the sections show hyperplasia of the endothelial cells of the blood sinuses and to such an extent that some regard it as a tumor. But these changes in the spleen of interstitial increase and hyperplasia of the follicles and of endothelial cells occur in varying extents in all of the spleen group.

A Physician: To what extent does the presence of ascites differentiate between the two conditions?

Dr. Richardson: I thought at one time that I had a method which would differentiate between the kinds of cirrhosis,—that one had ascites and the other had icterus. Unfortunately there are cases that have both, and I gave it up.

BOOK REVIEWS

Infection, Immunity and Inflammation.—A study of the Phenomena of Hypersentitiveness and Tolerance and Their Relationship to the Clinical Prophylaxis and Treatment of Disease, by Fraser B. Gurd, B. A., M. D., C. M., F. A. C. S., Montreal, Lecturer in Applied Immunology and Surgery, McGill University; Associate Surgeon, Montreal General Hospital; Consultant in Surgery and Surgeon in Charge, St. Anne's Hospital, Department of Soldiers' Civil Reestablishment; The C. V. Mosby Co., St. Louis; 1924.

Dr. Gurd writes simply and plainly. He takes a subject that has an endless number of apparently conflicting facts and observations, and offers a few theories, and most of the facts and observations fall into line; those that do not, seem relatively unimportant. This statement is probably like his theories—only relatively true.

He explains allergy, anaphylaxis and tolerance, and connects them all up with immunity in what appears to be a perfectly sensible way. Perhaps I can present a brief resume of his idea:

When foreign protein is injected for the first time into the tissues of an animal there comes about, slowly and gradually, illness in the animal. Nothing happens perhaps for hours or even days. Gradually the body is caused to form an antibody or a ferment which causes partial disintergration of the protein; a tthis stage it reaches somewhere near that of peptone; the protein then is toxic to the body. If sufficient protein was given the first time or, better yet, several moderate doses were given at short intervals, the body produces a considerable quantity of the antibody described above and called by Gurd "antibody of the first order." This antibody seems also to be the opsonin of Wright and not only stimulates leucocytes to ingest bacteria but actually to induce leucocytosis.

When a second injection of the protein is given after about two weeks, a marked local reaction may take place. This is the allergy of Von Pirquet, observed first in connection with tuberculosis. Allergy comes from two words and means altered reaction.

When the second injection of protein is given, the antibody present produces rapid disintegration of the proteins to about the peptone stage and, in case of allergy sufficient of them remain localized to cause inflammation. But if the toxic protein is absorbed rapidly, a more or less general toxic effect is produced. This is known as anaphylaxis. The state of the individual subject to either allergy or anaphylaxis is hypersensitiveness.

An individual hypersensitive to bacterial protein is immune to bacterial invasion, because his tissues destroy the protein of the bacteria. Such is the result of prophylactic bacterial vaccines.

The antibodies of the first order, it seems, may become saturated with the protein, and hence the individual is temporarily desensitized. This part of the theory is held by other writers.

The protein molecule which is left after being worked upon by the antibodies of the first order, stimulates the body to produce another antibody to produce further degradation of the protein molecule. These antibodies are designated "of the second order." They are best produced by repeated small injections of the whole protein. When those of the second order are produced in sufficient quantity, the protein is completely destroyed by being attacked first by the antibodies of the first order, and in rapid turn by those of the second order.

The agglutinin and lysins, as well as the opsonins, seem to be the antibodies of the first order, or to be closely associated with them. When the antibodies of the second order exist in quantity sufficient to destroy the peptone stage of the protein as rapidly as produced by the antibodies of the first order, the person is said to be in a state of tolerance, or is tolerant.

Antibodies are active only in the presence of complement. This brings in another factor of variability.

Antibodies seem to be the property of tissue cells and yet an animal may be made passively anaphylactic by transference of blood from a hypersensitive animal to a normal animal. Another strange fact is that, some hours after transference of the blood, the animal is more highly anaphylactic than at the moment of transference.

There are a number of facts not explained by Gurd's theories. He discusse not only allergy, anaphylaxis, tolerance, etc., but also all phases of im-

munity and inflammation. He believes much in vaccine therapy but he cautions against bacterial vaccine in the presence of acute infection.

Any physician can spend a few profitable evenings with this work.

Recovery Record—For Use in Tuberculosis. By Gerald B. Webb, M. D., Consulting Physician, Cragmor, Glockner, and Sunny Rest Sanatoria; Late Lieut. Col. M. P., U.S.A., Senoior Consultant A.E.F.; Former President National Tuberculosis Association; President Colorado School of Tuberculosis, Colorado Springs, Col.; and Charles F. Ryder, M. D., Cragmor and Glockner Sanatoria; Colorado School of Tuberculosis, Colorado Springs, Col.; second edition, revised; Paul B. Hoeber, Inc., New York; 1925.

This is not a large book as to size; its purpose, conception, and execution are colossal, and beautiful. The 79 pages of advice to the patient, preceding the blanks for the recovery record, constitute a wonderful appeal to the patient to put forth his every effort to recover his health. The language is used most artistically and effectually. We quote selected sentences: "Recovery from Tuberculosis is an adventure, with life itself as the goal of success." "Perhaps it is stupid and even insulting for the well to tell the sick, or the happy to tell the unhappy, to be cheerful."

One physician told his patient: "You have not more than one chance in five of recovering." The other said: "You have a good chance to get well, at least one chance in five!" Are not these two sentences worth any physician's time to read?

The book is worth the price for entertainment alone. The value for those who need it cannot be paid in cash. All tuberculosis patients should have copies. Physicians, recommend it to your patients with tuberculosis, and do both your patients and yourself a good turn.

Old and New Viewpoints in Psychology. — By Knight Dunlap, Professor of Experimental Psychology in the Johns Hopkins University; The C. V. Mosby Co., St. Louis; 1925; $1.50.

This volume of 166 pages presents five lectures entitled: Mental Measurements; Present Day Schools of Psychology; Psychological Factors in Spiritualism; The Psychology of the Comic; and the Reading Character from External Signs.

The reviewer lays no claim to special erudition along the lines of psychology. To psychologists he is a more layman. A layman's criticism must be taken only for what it is worth. With that apology I may make a perfectly worthless statement; Psychologists take themselves to d—— seriously. Professor Knight is a mild offender only; I read every word of his book. I enjoyed it. I may even read it again. I learned from it and I read it again I shall certainly pick up points I overlooked.

The last sentence of the book reads: "In the meantime, in the interests of the gullible public as well as the interests of psychology, both pure and applied, we must carry on an educational campaign against character analysis.

Psychologists re always studying reactions. Professor Knight will be interested mildly in the reviewer's reaction to his book and to know that he recommends it t oall who are in any way interested in the study of psychology.

Surgical Clinics of North America, for the year 1925, issued serially, one number every other month. Volume V, Numbers 1 to 6. Total pages 1704 with an average of about 125 illustrations in each number. Per clinic year, Paper $12.00; Cloth $16.00 met. Philadelphia and London; W. B. Saunders Company.

Chief interest in the Clinics for 1925 lies in the Philadelphia number which covers the meetings of the American College of Surgeons, and the Mayo clinic number. The others contain the usual run of good or average cases with here and there a mention of new technic, good procedure or lucid analysis. While we all desire to keep posted on rare and unusual cases, these can hardly be said to hold practical interest compared with the conditions that we meet and upon which we must pass judgment daily.

The college number with a symposium on gall bladder, Frazier on neurological surgery and Deaver on almost everything, holds first choice. Little incidentals such as, "Morphin never relieves the paroxysms of major trigeminal neuralgia; "Chronic appendicitis is a clinical entity and a surgical fact and not a fancy," and description of the on-coming blindness of pituitary disorders, thrown in with the consideration of some major condition revives the memory or recalls some forgotten case. Mayo, Judd and Balfour, with others not so well known, furnish a number that may be somewhat technical from a scientific standpoint but is certainly more interesting than much of the Mayo annual clinic volume.

E. B. R.

The Surgical Treatment of Goiter by Willard Bartlett, A. B., A. M., M. D., D. Sc., F. A. C. S., St. Louis; with foreword by Dr. Charles H. Mayo, Rochester, Minn.; with 130 original illustrations; The C. V. Mosby Co., St. Louis; 1926; $8.50.

There are books and books just as there are doctors and doctors. There are those of each who inspire confidence, and those who fail, to a greater or less degree, to inspire confidence.

Bartlett (with the publisher) has produced a book on the surgical treatment of goiter which impresses the reader that it is authoritative, that it is complete to the moment of leaving the author's hands and that it is most practical and usable.

The author's chapter on historical data is short, perhaps too short, but highly interesting. The reviewer at any rate could have read with pleasure many more pages of Dr. Bartlett's beautifully presented historical material. The chapter on Pathology is written by Louis B. Wilson of Rochester, Minn.; that on the Heart is by Samuel B. Grant of St. Louis; that on Laryngeal Complications is by French K. Hansel. All three chapters are highly creditable.

Bartlett's chapters include a discussion of unusual manifestations of goiter, indications for surgical treatment, preparation of the patient, types of operations, anesthesia and anesthetics, detail of operating, complications, after treatment, recurrences, etc.; these are just the subjects on which the surgeon wishes to get the words of the master. The reading of the chapters on technic would show the reviewer that the author is a master technician were he not already apprised of that fact.

Mr. Wm. French, who illustrated the book, deserves special mention. His illustrations are not just beautiful; they illustrate. Each illustration seems to have had study so that no more than necessary to convey the thought is pictured. There are 330 illustrations.

Bartlett could have written the book from his own experiences—so broad an experience has he had—and it would have been an excellent work. His familiarity with the accomplishments of other workers in the field, his knowledge of the literature and particularly his delightful use of language—make the book a valuable contribution. Surgeons will generally wish it on their shelves. We recommend it to them.

General Bacteriology, 8th Edition. Edwin O. Jordan, Ph. D.

This book needs no introduction to the bacteriologist or technician. It is very extensive and thorough. It takes in the usual procedure pertaining to staining methods, preparation of, and titration of, culture media by the newer Ph method and the old titration method, and all things pertaining to bacteriology.

The section on Pathogenic Organisms, their cultural characteristics, morphology, mode of dissemination and the epidemiology is especially good.

This book can be well recommended to the beginner as well as to the experienced worker.

EL PASO PERSONALS

DR. JAMES VANCE spent the holiday season in Mexico City, where he took part in the first amateur open golf championship of Mexico. Dr. Vance was runner-up in this tournament, losing the championship, to Claude Butlin, 3 to 2 in the final 36 hole match.

The wife of DR. J. N. STRATTON of Safford, Arizona, was operated upon in El Paso, December 12th. She is convalescing rapidly at this time.

DR. F. T. HOAGLAND, of Cananea, was in El Paso the latter part of January, en route to Mexico City.

DR. M. G. PADEN, of Carrizozo, was a professional visitor to El Paso, January 18th.

DR. R. E. McBRIDE, of Las Cruces, was a professional visitor in El Paso January 20th.

DR. F. O. BARRETT is doing post graduate study at Tulane University in New Orleans. He is limiting his study to pediatrics.

DR. AND MRS. JOE HILTON, of Mexico, were holiday visitors in El Paso.

DR. L. L. WITHERSPOON, who has been quite ill at William Beaumont General Hospital, is convalescent at this time.

DR. J. HAL GAMBRELL, who has been ill at the Masonic Hospital, is recovering rapidly at this time.

DR. FELIX P. MILLER returned January 28th from Tulsa, Okla., where he opened the discussion of a paper at the Sectional Meeting of the American College of Surgeons. The Section will meet in Little Rock, Ark., next year. Texas, because of its size, has asked for a separate meeting of its own, which Dr. Martin promised to arrange for next year.

DR. E. O. EARNHEART, gynocologist of Oklahoma City, is spending the winter in El Paso. He made a very interesting talk for the El Paso County Medical Society, January 31st. He spoke very highly of the climate of the southwest, stating that, if anything, it was underestimated by southwestern men as a resort.

DR. VICTOR E. BONELLI, of Fort Worth and Los Angeles, was a visitor at the County Society January 17th. He urged a Medical Arts buliding for El Paso, and cited Fort Worth's as an example of one that is a success in every way. He spoke highly of El Paso's climate as a rival of California's.

DRS. RILEY M. WALLER and R. B. McKNIGHT of Rochester Minn., have opened offices in the Two Republics building and will limit their work to surgery and surgical diagnosis under the firm name of Drs. Waller and McKnight. Both took their fellowship at Mayo Clinic. They are welcome additions to the El Paso profession.

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ARIZONA STATE MEDICAL ASSOCIATION, YUMA, APRIL 21 TO 23.

The General Program Committee, Dr. Charles S. Vivian, of Phoenix, Chairman, wishes to issue the positive declaration that the scientific program for this year's meeting will be closed thirty days in advance of the meeting, and will be published in the March issue of SOUTHWESTERN MEDICINE, which will be in the mails before March 25th.

Members of the profession in Arizona who have been in the habit of delaying the forwarding of titles of papers until the last moment will very likely find themselves without a place on the program. It is the desire of the Committee to use all the papers from Arizona which are available, but the titles and abstracts of papers must be sent in immediately, if they are to appear on the program.

Those who are planning to attend should, also, heed the advice issued last month and secure hotel reservations immediately. The Hotel del Ming will be the headquarters. This is a very excellent hostelry but is small and its capacity is very likely to be taxed to the uttermost by this convention.

NEW MEXICO MEDICAL SOCIETY

The annual session for 1927 will be held at Carlsbad on the 9th, 10th and 11th of May. Letters have been mailed to the secretaries of the various county societies of the state asking that they furnish papers for the scientific section of the program. It is to be hoped that not one of the county societies will fall down on this request and that we may have a fair representation of all the societies on the program.

In the January issue of SOUTHWESTERN MEDICINE request was made that members should, at once, get in touch with the secretaries of their societies and settle their annual dues and that members at large, affiliating only with the state society, should forward their checks direct to me.

By heeding that notice, as well as this one, much trouble and expense to the society will be averted and the chances of being dropped from membership on account of NON PAYMENT OF DUES much lessened. Some members, immediately upon reading the notice in the January issue, mailed checks for the annual dues of five dollars. Others have neglected this important duty.

Please do not delay longer, but right now, while fresh in mind, PAY YOUR DUES.

C. M. YATER, Sec'y.
Roswell, New Mex.

TEXAS STATE MEDICAL ASSOCIATION MEETS IN EL PASO

El Paso is busy preparing for the Convention of the Texas State Medical Association, which is to meet here April 26, 27 and 28. The formal program promises to be unusually good. There is an attractive list of distinguished guests.

The local men are preparing clinics and pathological exhibits of both fresh and preserved specimens that will prove unusual for a state meeting.

Entertainment, both domestic and foreign, is being planned by local committees and the work is sufficiently under way that it is safe to prophesy that this will be El Paso's premier accomplishment in the way of medical reception.

El Paso is hopeful of attracting a large

number of visitors from New Mexico, Arizona and Mexico, that her "close friends" may mingle with her "down state acquaintances."

Hotel reservations can be made at this time by communicating with Chairman of the Hotels Committee, Dr. Orville Egbert.

In connection with the meeting, the County Society is putting on a handicap golf tournament open to the profession of Texas, New Mexico, Arizona and Mexico, players attested by their local club authorities. Medal play will prevail, cards for 36 holes being turned in before noon of the last day of the session. The full 36 holes may be played in two days, or one day of play, cards to be turned in to the El Paso Country Club manager, properly signed and attested. Silver cups will be awarded the winner, two runners up, besides a number of minor prizes, such as clubs, balls, etc., therefore, all players should turn in their cards. Your badge will admit you to the course. Caddy fees are seventy cents for eighteen holes.

Cars will leave the Paso Del Norte at 5 a. m., 6 a. m. and 7 a. m. daily, or you may go out in a taxi at your convenience. The course is eight miles up the Valley Turnpike. The following members of the Golf Committee will take pleasure in seeing that you visit the links: B. F. Stevens, Chairman; Ralph Homan, J. A. Picket, E. D. Strong, James Vance, F. D. Garrett, George Bruner, C. P. Brown.

Dr. W. W. Waite is Chairman of the Scientific Exhibits for the State Medical Association and desires to invite our friends of the Southwest to prepare any exhibits, or demonstration they may have available.

WERE WE GYPPED?

In connection with the Basic Science Bill, recently before the Arizona Legislature, many members of the Senate had given the friends of this legislation reason to believe that they (the senators) were ready to vote for it. For that reason, it was re-introduced after being indefinitely postponed.

We had reason to believe that at least three members of the Public Health Committee of the Senate (Senators Lyons, Wills and Wylie) were prepared to report favorably on this bill. When it was reported unfavorably by this Committee, Senator Lyons excused himself to his constituency in Gila County by the statement that he was the only member of the Committee who was willing to report favorably. Physicians of Pinal and Cochise County who were so positive that Senators Wills and Wylie were in favor of this bill are asked to take notice of this statement by Senator Lyons.

As the matter now stands, there seems to be only one clear-headed champion of public health in the Senate, and that is Senator Bettwy of Santa Cruz County. This bill is clearly a public health matter, intended to protect the public from exploitation by unqualified practitioners of healing arts. The medical profession of the state, who undertook to introduce this legislation and explain its purpose, have either failed to get the message across, or else the legislative ears have been tuned to a different and less worthy wave length.

MARICOPA COUNTY (Ariz.) MEDICAL SOCIETY MEETINGS.

(Nov. 13, 1926)

Held at the Deaconess Hospital, Dr. J. E. Drane, president, in chair.

Dr. Schwarz gave a report of the Committee on Children's Week, explaining the different features of the clinic for the examination of children, and the plans of the clinic were discussed in detail. The age limit was fixed at six years.

Dr. Drane gave a report of the Committee on Sanatorium Situation.

The name of Dr. J. A. Ollerton, of Mesa, was reported favorably by the Board of Censors, and he was unanimously elected to membership.

Dr. George W. Stephens, of the State Hospital, gave a paper on "General Paralysis of the Insane," reporting his experience in the treatment of these cases, mentioning particularly the newer work with malarial infection. The subject was discussed by Drs. Clohessy, Sult, Hicks and Stroud.

Mrs. Marie Phelan, of the Federal Childs' Welfare Bureau, Washington, D. C., gave an interesting talk on the early milk feeding of infants as instituted by the public health departments of various cities throughout the United States since 1906.

(Nov. 27, 1926)

Held at St. Joseph's Hospital, Dr. Drane, president, in chair.

Dr. T. E. Schwarz, chairman of the Children's Clinic Week, held under the auspices of the Society, on November 18th, 19th and 20th, at the Chamber of Commerce in Phoenix, gave a report of the clinic. It was carried unanimously that a vote of thanks be extended to Mrs. Charles R. Howe, of the State Board of Health, to the Chamber of Commerce, the State Nurses' Association, the Maricopa County Dental Society, the Central Arizona Light & Power Company, the Deaconess Hospital, St. Joseph's Hospital, and other organizations which assisted in carrying on the clinic.

It was voted that the meeting night of the Society be changed from the first and third Saturday to the first and third Monday of each month.

It was voted that the second Monday evening of December be made ladies' night and the Ladies Auxiliary be invited to join with the Society in the annual meeting.

Dr. Sweek brought up the fact that the American Medical Association has gone on record as opposing the Sheppard-Towner bill; he also stated that some thought should be given to the matter of a new medical practice act before the coming legislature. It was voted that a program be ar-

ranged to discuss these matters and that Dr. Fahlen be asked to present the matter of the Sheppard-Towner bill.

Dr. Victor Randolph presented a paper on "Treatment of Pulmonary Hemorrhage," which was discussed by Drs. Phillips and Bannister.

Dr. Charles S. Vivian read a poper on "Concerning Urological Diagnosis," which was discussed by Dr. Sweek.

Both of these papers have been read before the Medical & Surgical Association of the Southwest, and will appear in SOUTHWESTERN MEDICINE in due course.

(Dec. 6, 1926)

Held at the Deaconess Hospital, Dr. Randolph presiding in the president's absence. Dr. Ollerton acted as secretary.

Dr. F. T. Fahlen, Superintendent of Public Health for Arizona, presented the matter of the Sheppard-Towner Act, its purposes and its administration in Arizona under his supervision. He considers that it has done a valuable work in Arizona and favors its continuation. During the past two years the work has been curtailed because the Arizona legislature failed to make the appropriation to match the Federal Aid. Many questions were asked and some criticisms were brought forth, all of which were answered by Dr. Fahlen.

Dr. W. W. Watkins presented a carefully prepared summary on hte "Deficiencies of the Arizona Medical Practice Act," with discussions by Drs. Garrison and Sweek.

It was voted that the Society take steps to secure a meeting of the House of Delegates of the Arizona State Medical Association, as soon as possible, to consider drafting an entirely new medical practice act to be brought before the January meeting of the legislature.

(December 20, 1926)

The annual meeting, with dinner for the members and the Ladies' Auxiliary was held at the Arizona Club, with about eighty present.

Dean Lane, of Trinity Cathedral, gave an after dinner address on "Spiritual Health in Relation to Physical Health."

Following the dinner, the ladies adjourned for entertainment at bridge, while the Society conducted its annual business session.

Dr. O. H. Brown, of the Committee on Radio Broadcasting, reported that a number of short talks have been prepared and that these have been edited by members of the committee.

Dr. W. W. Watkins, of the Committee on Newspaper Publicity, reported that the matter of using the available space in the Arizona Republican was at an impasse, owing to the requirement by the newspaper that the articles be signed, and the refusal of the Society to sanction this.

Dr. Randolph, of the Committee on Library, reported that space was available in the Carnegie Library for books and periodicals. Several questions were raised relative to the ownership of the books and periodicals if placed in the library and the care they would receive. The committee was asked to investigate further and report later.

The Board of Censors reported favorably on the application of Dr. Harry E. Braun, and he was elected.

Report of the Secretary-Treasurer was read, and its adoption voted.

The election of officers resulted in the following selections:

Dr. J. M. Greer, President.

Dr. W. A. Schwartz, Vice-President.

Dr. Victor Randolph, Secretary-Treasurer.

Dr. H. B. Gudgel, member of Board of Censors to succeed Dr. Fred Holmes, retiring member.

(January 17, 1927)

The first meeting of the year was held at the Deaconess Hospital, with the president, Dr. J. M. Greer, in the chair.

Dr. Greer gave a very lucid address on "The Function of the County Medical Society in Relation to its Members."

Dr. D. F. Harbridge read a paper on "The Relation of the County Medical Society to the State Association and the American Medical Association.

Dr. H. B. Gudgel read a paper on "A Review of American Medical Ethics."

In the discussion, the fact was brought out that the Society had once voted its approval of a consultant assuming charge of a case, on request of the patient, and regardless of his consultation relation with the attending physician. Upon motion, the Society voted to rescind that action. It was further voted that "any physician calling a consultant may exercise his right to ask the consultant to take charge of the case if for any reason, he so wishes."

Dr. R. E. Bradley, of Roswell, N. M., a visitor, was called on by Dr. Greer for a short talk and spoke very interestingly of his experiences with county medical work and medical ethics in New Mexico.

EL PASO COUNTY MEDICAL SOCIETY MEETINGS

January 17th

DR. R. L. RAMEY read a paper titled "Fractures of Elbow and Forearm"—a paper that he had previously read before the Southwestern meeting at Tucson. The paper will be published as part of that program.

In the discussion of the paper DR. E. J. CUMMINS emphasized that forearm fractures were emergencies and should be treated as such; should be set with the aid of x-ray and fluoroscope. He further stated that care must be taken to preserve the ossifaction centers in children to insure proper growth of bone.

DR. J. W. CATHCART stated that in his years of observation of fractures there is markedly higher percentage of good results in late years, which he attributed to dressing in the flexed position, plating and setting under the fluoroscope.

DR. E. B. ROGERS considers fracture of both bones of the forearm the worst with which the surgeon has to deal, viewed from a point of good results.

In closing, Dr. Ramey reiterated his position that dressing such fractures in the flexed position will give the best results. Even the fractures of condyles, if they are first reduced, then the arm placed in a state of flexion, the fragments will invariably remain properly aligned.

DR. G. WERLEY reported a case of aortic insufficiency in a man seventy years old, in which the Mayo Clinic, two years ago, had reported heart and aorta negative. Dr. Werley doubted syphilis being the etiological factor in all cases of aortic insufficiency and believed this case substantiates his belief, since the possibility of so old a man contracting syphilis in later years is very remote.

DR. E. B. ROGERS reported a case of dislocation of the acromio-clavicular joint, in which he was able to hold it reduced without an open operation until healed.

The Society unanimously endorsed the candi-

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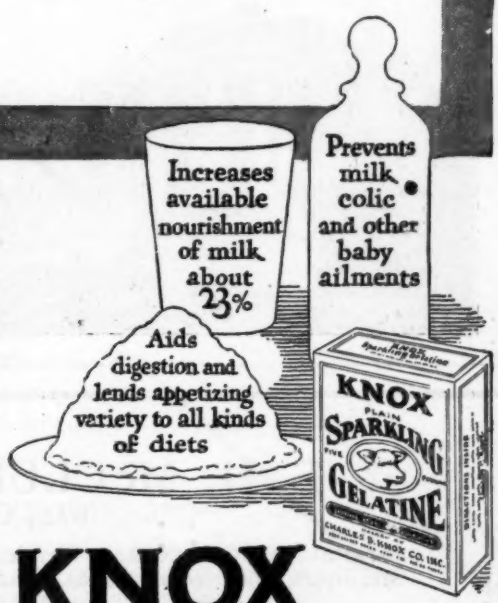
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January 24th

DR. K. D. LYNCH gave a lantern slide demonstration of non-calculus ureteral obstruction; his discussion was extemporaneous. The data compiled, together with the very beautiful illustrations will go to make up a paper which Dr. Lynch will present to the New Mexico Medical Society. It will, therefore, appear in SOUTHWESTERN MEDICINE at a later date.

January 31st

Dr. K. D. Lynch reported a case of Dr. Vance's in which he was associated. The case was apparently that of acute appendicitis, but in a couple of days developed acute suppression of urine, associated with a temperature of 102°. Within a few days the temperature subsided, together with the suppression, and repeated examinations of the urine were negative. The case was dismissed from the hospital. A few days later the patient returned with a pain in the region of the right kidney; the patient was cystoscoped and the urine and roentgenograms were negative. As the acute condition subsided there was a small hard mass palpable at the lower pole of the kidney. This gradually softened and disappeared, the patient apparently making a complete recovery. Diagnosis was acute abscess of the kidney, possibly attributable to a frontal sinus infection.

February 7th

DR. E. A. DUNCAN presented a case of diabetes in a child eight years old, requiring rather large doses of insulin to handle sufficient carbohydrates. The two points emphasized were that children require proportionately higher caloric food value than adults, and higher protein content in the diet.

In discussion of the case DR. J. A. RAWLINGS emphasized the value of insulin in these children since by its use, the patient can live practically, a normal existence. Prior to the discovry of insulin these cases always terminated fatally.

DR. HARRY LEIGH, in speaking of the control of these cases, brought out that in former days when they were managed only by diet, the child would occasionally go on a carbohydrate debauch, often resulting in acidosis, and even coma. Since the use of insulin makes possible a practically normal consumption of sweets, they are much more easily controlled.

In closing, Dr. Duncan emphasized that with insulin management of the diabetic child, it could lead a perfectly normal life.

DRS. W. L. and C. P. BROWN presented a paper on "Hematoma in Fractures" with an illustrating case, which will appear in an early issue of SOUTHWESTERN MEDICINE.

EL PASO CITY-COUNTY HOSPITAL STAFF MEETINGS

At meeting of the Staff of the El Paso City-County Hospital Dec. 15, 1926, the following officers were elected for the coming year:

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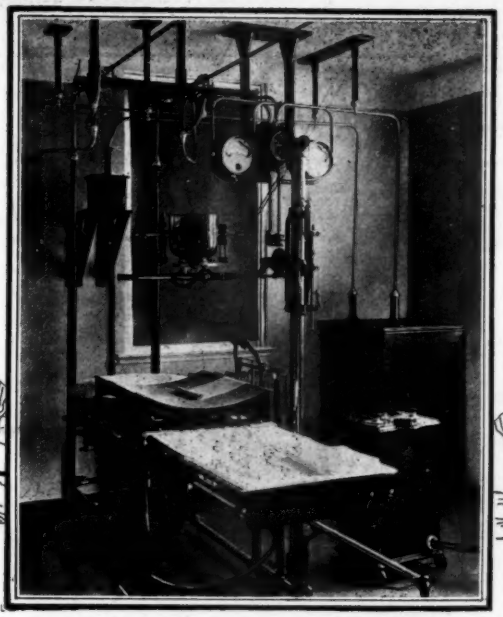

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Chairman of the Staff, Dr. R. J. Cummins;

Vice-Chairman of the Staff and Chairman of the Medical Service, Dr. G. Werley;

Secretary, Dr. L. M. Smith;

Chairman of Efficiency Committee, Dr. E. D. Strong;

To fill place on Efficiency Committee vacated by Dr. Strong, Dr. F. P. Miller.

The Staff of the El Paso City-County Hospital met following dinner at hospital at 7 p. m., January 19, 1927. The meeting was called to order by Dr. E. J. Cummins, chairman of the Staff. Those present were: Drs. McCamant, Miller, Strong, Richmond, May, Jamieson, Gallagher, Varner, Mason, Stevenson, Will Rogers, Long, Cathcart, Nell, Outlaw, Barnes, Werley, Thompson, Armistead, Rigney, Cummins and Smith.

There being a custom that deaths occurring during the month are discussed by the staff members on whose services they occur, the following cases were discussed:

DR. THOMPSON reported a death from gastric carcinoma, and another from fractured cervical vertebrae. The latter was sent to the hospital for a fractured jaw, and the broken neck was discovered at autopsy.

DR. WERLEY reported a death on his service from diabetes, and one from angina pectoris with decompensation and mental symptoms. He emphasized the fact that mental symptoms are common in heart failure, and are not due to the digitalis therapy as sometimes thought. Autopsy of this case showed arteriosclerotic kidneys, aneurism, thrombus of right coronary artery, and obliteration of the left coronary artery.

DR. LONG reported the death of a patient with pulmonary and laryngeal tuberculosis. Pneumothorax was performed. On account of the laryngeal involvement the patient had extreme difficulty in eating.

DR. STEVENSON discussed a death from a bullet wound of the abdomen which penetrated the right lung. Autopsy showed a hemorrhage into the lung.

Dr. Stevenson also reported a death from intestinal and peritoneal tuberculosis with adenocarcinoma of the omentum. The patient had great distention and absolute obstruction. The omentum was extremely brittle. The bowels were studded with miliary tubercles. Enterostomy was performed for relief of symptoms, but the patient died one week after operation. There were two gallons of fluid in the peritoneal cavity.

DR. VARNER reported a premature birth with placenta praevia; also a stillbirth with polycystic kidney.

DR. JAMIESON reported a case with a variety of pathology. The first symptoms had been frequent and painful urination, and pain along the course of the right ureter. Cystoscopic examination showed stricture of the left ureter. The right ureteral orifice could not be found. Operation at this time showed a small right kidney containing pus, an autonephrectomy. Later the patient developed pain in the left side. A gastrointestinal series of roentgenograms showed a spastic colon. The patient was put on a diet and the pain disappeared. She then developed a lump in the left breast the size of a walnut. Dr. Cummins performed a radical breast amputation. Pathological examination of the tumor revealed carcinoma.

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Later she had pains radiating down the right side. Pelvic organs were negative. The patient was admitted to the City-County Hospital Nov. 25th, with a boring pain in the abdomen. Operation showed a cystic ovary and tuberculous peritonitis. Pleurisy followed the operation, and the patient died three days later. Autopsy was not performed.

DR. ARNSON reported three deaths: one skull fracture, one lobar pneumonia, and one chronic endocarditis. These patients died soon after coming to the hospital, and were not seen by members of the visiting staff.

After the business of the staff was transacted hte meeting adjourned at 9:45 p. m.

L. M. SMITH, Secretary.

ST. JOSEPH'S HOSPITAL (Phoenix) STAFF MEETING

January 10, 1927.

The regular January meeting of the Staff of St. Joseph's Hospital registered the largest attendance of any hospital staff meeting so far held in Phoenix, exactly fifty members being present, with two Sisters from the hospital management.

The program was on Acute Appendicitis, reviewing the work of the past three years in this hospital on this class of cases. The following program was presented, the papers and discussions appearing elsewhere in this issue of SOUTHWESTERN MEDICINE, as a "Symposium on Acute Appendicitis."

"Statistical Report on Two Hundred Cases of Acute Appendicitis Operated in St. Joseph's Hospital During Three Years (1924-26)." W. Warner Watkins, Secretary of Staff.

"History and Etiology of Appendicitis," Dr. J. M. Greer, Mesa; Discussion by Dr. S. D. Little, Phoenix.

"Symptoms and Diagnosis of Acute Appendicitis in Adults," by Dr. H. B. Gudgel, Phoenix; discussion by Dr. O. H. Brown, Phoenix.

"Symptoms and Diagnosis of Acute Appendicitis in Children," by Dr. Dudley Fournier, Phoenix; discussion by Dr. John Wix Thomas, Phoenix.

"Pathology of Acute Appendicitis," Dr. H. P. Mills, Phoenix; discussion by Dr. H. L. Goss, Phoenix.

"Anesthesia in Acute Appendicitis," Dr. S. I. Bloomhardt, Phoenix.

"Treatment of Acute Appendicitis," Dr. E. Payne Palmer, Phoenix; discussion by Dr. A. M. Tuthill, Phoenix.

Following the general discussion, it was announced that the February meeting would be based on the obstetrical work in the hospital during the past two years, and the program would be in charge of Dr. Kimball Bannister, the member of the Executive Committee in charge for February.

W. WARNER WATKINS, Secy.

DEACONESS HOSPITAL (Phoenix) STAFF MEETING

The Medical and Surgical Staff of the Arizona Deaconess Hospital met Monday evening, Jan. 24th, with 27 members and associate members in attendance.

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The Records Committee's report upon the December deaths was made by the secretary:

Case 6874; male, 46 years of age, chronic pulmonary tuberculosis; probably also tuberculosis of the bowels and chronic nephritis.

Case 6896; male, 48 years old. Perforated gastric ulcer; he had a previous perforated gastric ulcer a year before, was operated upon and recovered. He was again operated for this second perforation, which was in a different location. Admitted Nov. 13th, and died on Dec. 11th.

Cases 6914 and 6915; mother and babe, eclampsia and premature birth at eight months.

Case 6946; male 66 years of age, with taboparesis. He was sent to the hospital in a dying condition and lived only a few days.

Case 6954; was a male, 55 years old, with myocarditis. Death came the day after admission.

Case 6979; was a male, 57 years of age, with hemorrhagic nephritis. Admitted Dec. 18th and died Dec. 27th.

Case 6980; a male, 43 years of age, admitted 26th and died the 28th. Diagnosis of acute nephritis, pleurisy with effusion, complicating an acute respiratory tract infection.

Case 2831: By DR. A. A. SHELLEY. Female child, two years old, of perfect physique, well nourished and healthy; had never been sick.

The child's mother had been to the Mayo's a few years previous (1920) where it was said that chronic sores in the mouth were caused by bad teeth. She promptly recovered after removal of the teeth. A Wassermann at the time was negative. The father had always been healthy, says he had never had venereal disease and a blood Wassermann made at this time is negative.

The child was first seen on Aug. 22, 1924 and the grandmother gave the following history. About nine o'clock of this day, it was noticed that she had some fever, although she was still playing and apparently feeling well. Her grandmother gave her two or three doses of castor oil which she promptly vomited. She became worse and when examined she had a temperature in axilla of 104, pulse 140, very coated tongue, abdomen slightly distended, and she was very nervous with twitching of the eye lids and some spasmodic muscular movements. Lungs normal. Heart sounds normal. There had been moderate bowel movement in the morning, and so far as known kidneys had acted normally. Attention was called to a very slight abrasion on the ball of the right foot evidently a slight hurt from running around in bare feet. A drop of pus had been expressed that morning. She had not complained of pain, but there was a slight enlargement of the inguinal glands of the right side.

Shortly after two o'clock she had a convulsion of short duration. About three o'clock she had another which was promptly relieved by warm bath in bath tub. Wound on foot was freely opened and curetted. Very soon there was another convulsion which did not end until given a dose of morphine and atropine. The temperature dropped to 101.6, pulse 130. Breathing somewhat irregular, but gradually improved. The child never regained consciousness after the first convulsion. About this time it was recalled that the child had a moderate diarrhea two days before; some urine was secured by catheterization; the child was removed to hospital. She rested

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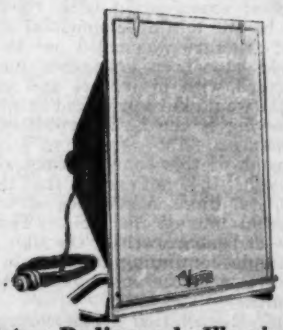

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quietly until about eight o'clock when she had another slight convulsion and the attending nurse had to resort to artificial respiration for a time.

Soon after the last convulsion, a spinal puncture was made getting only blood of which there was about four drachms; it coagulated at once. Dr. Watkins then made a spinal puncture getting a very bloody fluid, which proved to be free of pus or organisms. The blood gave a one plus positive Wassermann reaction. Complement fixation for tuberculosis one plus positive. The urine had a trace of albumen, and occasional pus and blood cell.

The child died about midnight of the same day.

In answer to questions, Dr. Shelley said that a definite diagnosis was not made, though it was felt that syphilis was a contributing factor. No blood culture was made.

Case 7022: By DR. E. H. BROWN. Male, age 15 years, well developed and nourished. Physical condition normal except left leg and foot.

About eighteen months ago while playing baseball at school, was struck upon the left ankle by the ball; this injury was very painful at first, but was not severely enough to make him stop playing. The greater part of the pain soon subsided. The ankle continued tender and sensitive for some time. A few months later it was noticed that there was a tendency for the foot and ankle to turn in; this condition became gradually more marked up until the present time. When walking he is unable to place the foot flatly upon the ground—the weight being taken on the outer side of the foot. At the present time there is a general weakness through the ankle but very little pain.

Examination shows a marked deformity of the lower third of tibia; the appearance was typical of a mal-united fracture of the tibia and fibula that permitted an inward deviation of the lower end of the leg, ankle and foot. No irregularities of the bones could be detected by palpation.

The left tibia measured three-fourth inch shorter than the right. There was one inch difference in the circumference at the calf of the legs. Ankle measurements showed three-eighths inch difference.

By physical examination the deformity was thought to be due to a developmental condition. The question of fracture was ruled out by the history, for it would have been impossible for this boy to have continued with his school and games with a fracture that would have produced this condition.

The following is the x-ray report of Dec. 26, by Dr. Watkins:

"Radiograph of this bone does not show evidence of new growth. It is not certain that there has been a fracture, but there is evidence of some lesion in the epiphyseal line of the tibia. This gives the appearance of bone growth of the tibia having stopped—the fibula continuing to grow in length, producing distortion at the ankle joint with rotation of the astragalus."

Diagnosis: Retardation of the normal growth of the lower end of the left tibia, due to epiphyseal injury. The growth of the fibula being normal, this condition produced a gradual bowing of the lower third of the tibia, thus changing the plane of the articular surface of the ankle joint by the growing fibula pushing the outer side of the foot downward and inward.

All long bones have an epiphysis at each end, with the exception of the metacarpals, the metatarsals, the phalanges of the hands and feet, and the clavicles, all of which have an epiphysis at only one end.

The increase in bone length takes place at the epiphyses. In the long bones, ossification always proceeds more rapidly at one one end than at the other and the growth is more rapid and continues longer at the end at which the epiphysis is last to unite to the shaft.

All through immaturity the epiphyses freely communicate with the diaphysis; in early life the epiphyses are completely shut off from the disphysis by the epiphyseal cartilages. The epiphyses obtain their blood supply from the periosteal net work of arteries—large branches of which perforate the thin layer of compact tissue on their exterior, and are distributed throughout the spongy cancellous tissue. Nearly the whole of the blood supply is therefore independent of the dipahysis. Only one or two minute arteries pass into the epiphyses from the diaphysis through the conjugal cartilage. This accounts for the comparatively infrequent occurrence of necrosis of the epiphysis even when the diaphysis is more or less completely displaced from the epiphysis.

Ossification takes place from the center of the epiphysis and proceeds toward the periphery until the entire epiphysis is ossified, with the exception of a thin layer at the junction of the shaft forming the epiphyseal cartilage or disk.

There is no doubt but that this boy suffered sufficient injury to the blood supply of the lower tibia epiphysis to cause this retardation growth, and resultant deformity.

Treatment: In order to bring the foot and ankle back to normal position a sub-periosteal osteomy was done through the lower third of the tibia, two inches above the epiphyseal line, as well as a resection of three-fourths of an inch of the fibula. This permitted a moderate over correction of the deformity; the foot was put in plaster in this position.

The reason for the over correction is that the boy will not reach his full growth for at least another three years, and hence there will be a tendency for a recurrence of the deformity.

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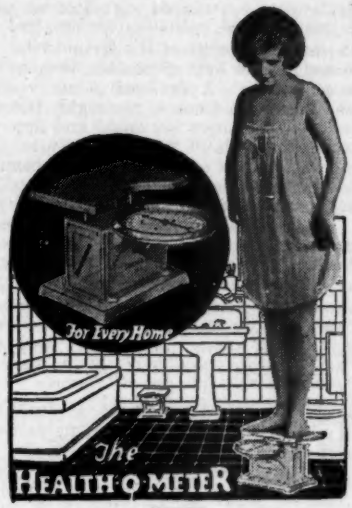

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The chances are that the normally growing fibula will cause a sufficient recurrence of the deformity to call for further operative interference. A resection of the epiphyseal end of the fibula may have to be done. The left leg will ultimately be from three-fourths to one and a half inches shorter than the right.

The secretary presented records of two cases of leukemia, summary of which follows:

Case 1923, female, Pima Indian, 32, admitted in drunken condition, with lacerations about head from a fight. The routine blood examination revealed 177,000 leucocytes with anisocytosis, poikilocytosis, microcytes, macrocytes and normoblasts. There were 11 per cent large lymphocytes, 52 per cent polynuclears, 22 per cent basophiles, and 15 per cent myelocytes. On these finds the diagnosis of myelogenous leukemia was made.

Four subsequent examinations showed counts varying from 145,000 to 250,000, hemoglobin from 30 to 45 per cent, red cells 2,350,000, with other findings as at the first examination.

This patient, supposedly healthy, except for superficial wounds, having been shown by the routine blood count to be an interesting medical patient, the secretary of the staff, in consultation with Dr. Dameron, made a complete examination with the following results:

Female Indian. About five feet, two inches tall, weight about 115 pounds. Chocolate colored skin; lips of fair color; wound on back of head; several slight abrasions on left side of face, about eyes. Eyes, dark, pupils round, reacting but sluggishly. Patient breathes freely through the nose. Palpable lymph nodes in neck present. Tonsils present and apparently diseased. Mucous surface of mouth is abnormally pale. Full set of teeth, two probably devitalized.

Chest: Well formed. Slight depression above and below the clavicle—little more noticeable on the right than on the left. Percussion shows slight dulness over left hilum. Definite sura-sternal notch pulsation is visible, and palpable. Mediastinal dulness extends up to upper edge of the second rib. Heart dulness 8 cm. to the left of median line, by percussion and auscultation. Apex beat is not visible. Right margin of heart is 4 cm. to the right. Blood pressure, 120/58. Radial pulses are equal and apparently normal. Heart sounds of normal intensity, but systolic murmur is heart at apex, slightly transmitted to axilla; murmur seems to increase in intensity in direction of second left intercostal space. Heard loudest about junction of the left fourth rib and sternum. Slight diastolic murmur heard, also in region of left fourth interspace at the edge of sternum. No axillary lymph nodes are palpable.

Abdomen: Normal in appearance, except for linea alba and median incision scar extending one and one-half inches below umbilicus to pubis. Liver dulness from fourth rib to the costal arch, in mamary line.

The spleen extends from the seventh rib in mid-axillary line to costal arch and is palpable just above the level of the costal arch.

Extremities: Left forearm shows extensive scar deep into flesh from elbow joint to within two inches of head of radius. Practically all of outer portion of muscle is destroyed. There is slight edema just above the right ankle, probably from slight wound on ankle.

Patellar reflexes are active and equal. Achilles and Babinski, Gordon and Oppenheim are normal. Posi-

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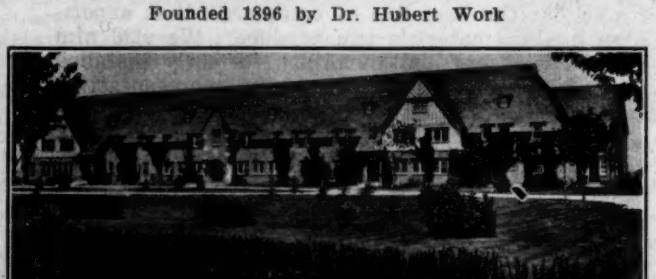

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tive Chaddock. Abdominal reflexes not obtained.

Vaginal exammination: Perineum shows posterior laceration. Cervix shows very marked laceration. Small uterus palpable. Slightly more resistance to the left of cervix than to the right.

Laboratory findings: Blood: See report of routine examination, above; the Wassermann reactions were strongly positive, Kolmer titration being 30 plus; the Kahn test was also positive. Complement fixation reactions for gonorrhea were positive; complement fixation reactions for tuberculosis were negative.

Diagnosis: Scalp wound. Wound of ankle. Scars from former injury of left forearm. Cardiac lesion, probably double aortic and mitral Dilated aorta. Syphilis; myelogenous leukemia. Lacerated cervix and perineum. Devitalized teeth.

Case 5325; male, age not given. Brought to the hospital in a condition unable to give history and no friends or relatives could be found.

Examination showed fairly well nourished, medium sized man, slightly cynanotic, lying restlessly in bed, apparently apprehensive. Slight icteric tinge to the conjunctivae; pupils equal, regular, and reacting to light and accommodation; no ptosis, nystagmus, or strabismus. No enlargement of the thyroid, but the posterior auricular lymph nodes are markedly enlarged, firm, easily movable, and about the size of No. 4 buck shot.

Apex impulse of the heart is seen and felt feeble in the sixth interspace, 12 cm. from the mid-sternal line. There is a soft blowing systolic murmur at the base, transmitted over all the pericardium. This murmur is probably functional. There is no bruit in the region of the aortic valve.

The right lung is resonant throughout. Expansion is good. There are no abnormal sounds. In left lung breath sounds indistinct but there are no rales except at the apex extending as low as the fourth interspace, where a few scattered coarse rales are heard.

Rounded margin of the liver is felt four finger breadths below the costal margin, tender but not nodular. There is an area of increased duiness to percussion in the left hypochondrium, which is apparently in front of the colon, and suggests the possibility of enlargement of the spleen. No definite enlargement of the spleen is felt.

The skin over the entire body is dotted here and there with hemorrhagic areas resembling purpura hemorrhagica. Bones and joints are negative, except that there are areas of periosteal thickening in shin bones suggesting the possibility of an old syphilitic periostitis Throughout the surface of the body are enlarged glands about the size of No. 4 buck shot, freely movable, and not adherent. Glands in the groin are noticeably enlarged, being the size of No. 3 buck shot.

The urine showed albumin, casts and acetone. The blood examination showed 90 per cent Hbg; color index 0.9 per cent; 4 970,000 reds; 24,600, whites with 18 per cent leukocytes, 80 per cent neutrophiles., 1 per cent eosinophiles, and 1 per cent basophiles. The Wassermann was one plus positive. Death occurred at the end of six days.

Autopsy by Dr. Watkins:

Fairly well nourished man, with numerous small superficial skin ulcerations, having hemorrhagic crusts. All the superficial lymph nodes enlarged, the inguinal group being most enlarged. Skin and muscles showed a yellowish tinge. In chest, right lung was normal. Left lung showed dense pleural adhesions. Heart not enlarged. No evidence of aortic enlargement or valvular disease. Anterior and posterior mediastinal lymph nodes much enlarged, soft and friable.

Liver much enlarged, not weighed. Showed innumerable discrete yellowish spots, those on the surface projecting as small granulomatous nodules. Throughout the cut surface the same appearance, the entire liver being filled with these miliary areas. Spleen enlarged, showing the same appearance as the liver, with the projecting nodules of about rice grain size, with entire substance filled with the discrete areas of similar size. Polycystic kidney on each side. All the lymph glands of the abdomen enlarged, soft and friable.

Sections of lymph nodes show extensive areas of softening with loss of cell outline and hemorrhagic extravasation. There are numerous areas of infiltration by atypical cells, resembling large lymphocytes. Sections of spleen show infiltration with cells as found in lymph nodes, this change being chiefly perivascular. Sections of kidneys show marked degeneration of epithelium of tubules, with extensive desquamation and with dilation of both convoluted and straight tubules, and there are numerous microscopic as well as macroscopic cysts. Sections of liver show extensive perivascular infiltration by large atypical cells, similar to those found in lymph nodes and spleen. There is a moderate amount of passive hyperemia presnt.

Microscopic Diagnosis: Leukemia with leukemic infiltration into liver, spleen and lymph nodes.

DR. H. P. MILLS opened the discussion on leukemia as follows: A systemic disease characterized by hyperplasia of the leukocyte producing tissues and accompanied by a secondary disturbance of the composition of the blood which manifests itself in

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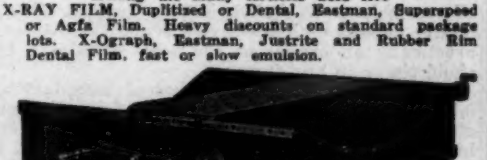

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alterations in the relative proportion of the leukocytes, the appearance of the forms not normally found in the total number of white cells.

Two general types are described, the myeloid or spleno-myelogenous and the lymphoid or lymphocytic. In the former the essential pathology lies in the spleen and bone marrow and the blood contains forms of granulocytes or myelocytes and there is associated disturbance of red cell production, with resultant anemia of varying degree. In the latter there is a great excess of lymphocytes in the peripheral blood and there is associated generalized hyperplasia of lymphoid tissues of the body.

In chronic myelogenous leukemia the spleen is usually enlarged, frequently to many times its normal size and on microscopic section shows extensive infiltration with bone marrow cells which largely replace the follicles, and the venules of the splenic pulp are filled with myelocytes, polymorphonuclear neutrophiles, mononuclear cells and nucleated red cells. The bone marrow shows cellular hyperplasia, has a pinkish color and the capillaries are filled with myelocytes and leukocytes of all kinds. The lymph nodes usually show only slight enlargement and microscopically may present little change. The blood changes are very marked. The total red count is low, except in early cases as is also the hemoglobin, but the color index is usually high. Nucleated red cells are commonly present and abnormally of size and contour of red cells is usually evident. The most striking feature of the blood picture is the great increase in the number of leukocytes. The count is usually above 100,000 and sometimes reaches 1,000,000. The differential count varies much in different cases but the presence of myelocytes in the peripheral blood is a prominent feature, the percentage varying from twenty to eighty.

In lymphatic leukemia the spleen is enlarged, but to a less degree than in the myelogenous type. The bone marrow has a pink or reddish color and the erythroblastic and myeloblastic tissue is largely replaced by lymphocytes. The lymph nodes over the entire body show marked hyperplasia and on section show almost solid masses of lymphocytes and loss of all resemblance to their original structure. This excessive deposit of lymphocytes is seen on section of practically all organs of the body containing lymphoid tissue in any form.

The blood picture is more that of secondary anemia, than is seen in the myelogenous form, the hemoglobin and red cells are both lowered and the color index usually low. Nucleated red cells occur, but not so commonly as in the myelogenous type. The increase is in the lymphocytes, these reaching a percentage of forty to ninety.

Rarely blood counts are found in acute infections involving the lymphoid structure, in which the differential count is reversed, there being a moderate leukocytosis with eighty to ninety per cent lymphocytes. These cases usually return to normal, but should be held under observation as possible early cases of leukemia and frequent counts made Occasionally there are periods in the course of lymphatic leukemia, during which the clinical findings remain unchanged, but the blood picture becomes practically normal; and rarely there are cases in which these findings persist throughout even to the death of the patient. To this group of cases has been given the term aleukocythemic leukemia. Case 5325 apparently belongs to this type. These cases have all the clinical and pathologic findings of lymphatic leukemia, except that the blood picture remains practically normal.

DR. O. H. BROWN presented Case No. 33 as follows:

Male, 56 years of age. He fell from a tree to the ground, a distance of perhaps 10 feet. He struck the ground on his back, perhaps touching the ground first with his buttock. The ground onto which he fell was a drive and hence was very hard. He was picked up by his family and neighbors and placed upon an army cot. Here he was found by the physician. He was pale; his body was cold and covered with free perspiration. His pulse however was good. He was complaining of severe pain in his back and abdomen and his breathing was jerky and accompanied with a grunt. The legs were paralyzed. The abdomen was large and somewhat tense, but no special areas of spasticity could be elicited. A working diagnosis was made of shock, fractured vertebra hemorrhage or other injury of the lower cord and a possible abdominal injury. A dose of morphine (one-fourth grain) strychin .1-30 gr.) and tropin (1-150 gr.) was given hypodermically.

When ambulance arrived the door would not admit the cot. The drivers were instructed to return for an ambulance with a rear door.

He was placed in the ambulance without being disturbed on the cot. At the hospital about a dozen hands were put under him and he was gently rolled over enough to slide under him a Bradford frame. He was carefully drawn by many hands into a comfortable position on the frame. By means of this frame he could be lifted to the x-ray table, to bed, and back again if necessary. It took several attempts to get satisfactory x-rays as he was a heavy man and the canvas and rope on the Bradford frame cast shadows.

After he was put to bed a more thorough physical examination was made. Skin was cold, pale and covered with perspiration; blood pressure was 90/60; abdomen was distended and generally tense; legs paralyzed; patellar, achilles and toe reflexes were absent; pin pricks and touch were not felt from two inches below the umbilicus in front and the twelfth dorsal vetrebra behind to the soles of his feet; heat and cold were not felt in the paralyzed area. The diagnosis at this point was the same as before. It

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was concurred in by consultant. Operation was thought inadvisable, at least until morning.

The x-ray, the next morning disclosed a fractured twelfth dorsal vertebra and a fractured rib near the spine. The signs of shock were still present, the bladder was full of urine which he was unable to void and evacuation of his bowels had not been obtained. Catherterization of bladder obtained urine with a trace of blood; this probably came from the catheter.

Operation was again postponed. Purges and enemas were given with ultimately slight results. His blood pressure came up under pituitrin and sodium coffein benzoate to 120/80. The spinal fluid was nearly pure blood and was scanty. Early in the morning of the fourth day he vomited a large amount of dark colored fluid that the night superintendent thought was fecal in character. This caused the operation to be postponed again.

From this on there was a gradual decline and he died near the end of the fifth day. He vomited a large amount of fluid just before death.

An autopsy was refused. The death certificate was signed paralysis of bowels, secondary to fractured twelfth dorsal vertebra and spinal cord hemorrhage.

At one time it was considered that there might be a ruptured bowel, but was finally considered improbable. It was also considered that an acute dilation of the stomach might have been an important cause of death.

DR. E. P. PALMER: Fracture of the bony structure of the spine is always a grave injury and its diagnosis, prognosis and management present serious problems to the surgeon. Fracture-dislocation usually produces a partial or complete separation of the sigments of the cord, so complete recovery is rare. Regeneration of the non-neurilematous elements of the cord does not occur, so, following a division of the cord between the atlas and the first lumbar vertebra, no improvement is to be expected except through extra spinal nerve anastomosis. There is always uncertainty regarding the pathological condition. The x-ray is of assistance in determining the injury to the bony structure, but unfortunately, the extent of the injury to the bony canal is no guide to the damage done the cord. There is but one safe course in all cases of spinal injury presenting deformity and paralysis, when the patient's condition will permit, that is, to perform an immediate exploratory laminectomy to determine whether operative treatment will improve conditions. We must remember that in all dural and bone work, our operative tecnique must be perfect. I wish to emphasize the importance of immediate operation if the patient's condition will permit, as delay is dangerous. The cord may be pressed upon by bony fragments or by a blod clot within or without the dura, which pressure, if relieved at once, will leave the cord uninjured, but if allowed to reamin a few days or weeks, will result in degeneration. It has been shown at many late operations that the only lesion to the cord was compression, but the late operation was of no avail as the degeneration had occurred. An early operation in such cases, would, of course, have relieved the pressure and prevented the degeneration changes. In the case under consideration there was a fracture-dislocation of the twelfth dorsal vertebra resulting in anesthesia and paralysis to the height of anterior superior spinous processes in front and to the point of spinal injury behind, resulting in retention of urine and feces. When the patient was first seen it was thought that he could be gotten in good enough condition to permit of laminectomy under nitrous oxide-oxgen novocain anesthesia and every effort was made to improve his condition with that object in view. Twice when he seemed improved the time was set for operation and the operating room was ready; each time symptoms arose which prevented us from undertaking operation.

DR. O. H. BROWN: Tilney and Riley say that the parastaltic action of the intestines seems to be stimulated by the vague and inhibited by the sympathetics. In case our diagnosis is correct the spinal cord lession was a stimulating one and caused over action of the sympathetics. Of course there may have been a thrombus in a portion of the intestines.

DR. TUTHILL said that the man must have had more than a fractured vertebra as such cases usually live about five months.

DR. MILLS said the twelfth dorsal is about the right limit for the sacral sympathetics.

DR. BANNISTER said he thought the question of paralysis of bowels from spinal cord pathology was particularly interesting to him as he had had two cases lately with spinal cord lesions both of which had to have bowels moved for them, and had serious results therefrom.

Adjourned. Orville Harry Brown, Sec.

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